STATE OF NEW MEXICO
BEFORE THE ENVIRONMENTAL IMPROVEMENT BOARD

IN THE MATTER OF PROPOSED REGULATION
20.2.350 NMAC – *GREENHOUSE GAS CAP AND TRADE PROVISIONS*
No. EIB 10-04 (R)

DIRECT TESTIMONY OF JONATHAN PATZ

Good morning Madame Chairwoman and other distinguished members of the Board. Thank you for the opportunity to appear before you in this hearing to address the public health impacts of Climate Change, a topic that I have studied for more than 15 years. I served as Co-chair for the Health Expert Panel of the U.S. National Assessment on Climate Variability and Change and have been a Principle Lead Author on five reports of the UN Intergovernmental Panel on Climate Change (IPCC) since 1995. I am a physician and a Full Professor at the University of Wisconsin at Madison, where I direct global environmental health research for the Nelson Institute, Center for Sustainability and the Global Environment (SAGE), and the Department of Population Health Sciences in the School of Medicine and Public Health. I have conducted extensive research and teaching in the field of environmental public health, specifically addressing global climate change.

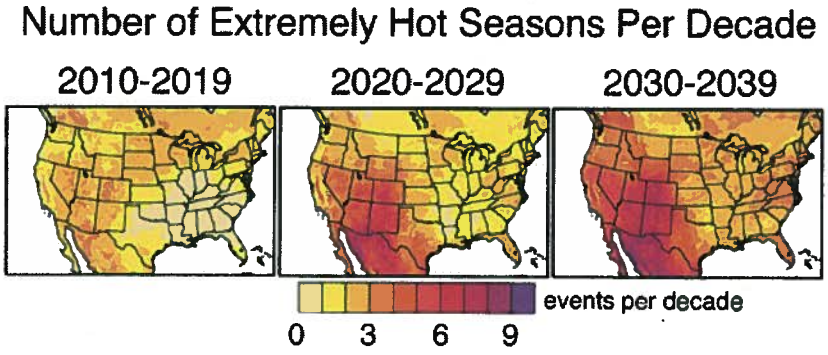
I. NATURE OF THE PROBLEM

Global warming is unlike many other health threats which we have confronted because unlike 'single agent' toxins or microbes, climate change affects multiple pathways for harmful exposures to our health. Climate change can affect human health

from direct heatwaves and severe storms to ground level smog/ozone pollution and airborne allergens, as well as many climate-sensitive infectious diseases.

Disease risks originating outside the U.S. also must be considered because we live in a globalized world. Many poor nations of the world are expected to suffer even more health consequences due to climate change compared to the U.S. With global trade and transport, however, disease flare-ups in any part of the world can potentially reach the U.S. Additionally, climate extremes, e.g. droughts and storms, can further stress environmental resources by destabilizing economies and potentially creating security risks both internally and from other nations.

Finally, while climate change is a long term environmental threat, the health ramifications are already occurring. Global warming in just the past 30 years may already be adversely affecting the global burden of disease. NMED-Patz Exhibit 1. And while single climate events cannot be attributed to climate change, more than 50,000 deaths in the 2003 European heatwave remind us of the risk of extreme weather events. Indeed, a study in *Nature* concluded that global warming over the recent decades doubled the 'probability' of the occurrence of such an extreme heat wave. NMED-Patz Exhibit 2. The implications for New Mexico are troubling. A new study finds that by the year 2039, most of the U.S. could experience at least four seasons per decade equally as intense as the hottest season ever recorded from 1951-1999. In most of Utah, Colorado, Arizona and New Mexico, the number of extremely hot seasons could be as high as seven per decade. The following maps demonstrate the rise in hot seasons.

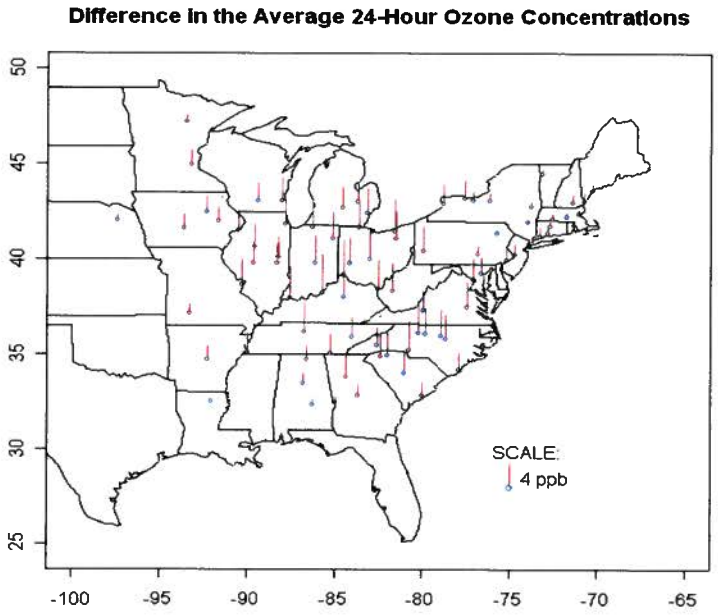


NMED-Patz Exhibit 3.

A. WHAT ARE SOME OF THE POTENTIAL IMPACTS OF CLIMATE CHANGE IN THE UNITED STATES?

Climate-related disease risks occur throughout the U.S., and many are expected to be exacerbated by climate change. Some health benefits could result, including reduced cold-related mortality and the incidence of Rocky Mountain Spotted Fever in the Southeastern U.S. However, the net health effects have been assessed to be adverse.

Our country has experienced deadly heatwaves (e.g, the 1995 heatwave killed more than 700 persons in Chicago alone), and according to climate models, heatwaves will become more frequent and intense. For example, Los Angeles is projected to experience a three-fold increase in heatwaves by the end of this century, while major portions of the U.S. are expected to have a higher number of extremely hot days. Preliminary analysis from my own research finds that the frequency of extreme heatwaves in Wisconsin will increase disproportionately compared to a smaller decline in the frequency of extremely cold temperatures. The poor and elderly populations are especially at risk of dying in heatwaves.



Air pollution accompanies heatwaves, due in part to the temperature sensitivity of the chemical reaction that forms ozone smog pollution. A recent study of the 50 largest cities in the Eastern U.S. finds that by mid-century, 'Red Ozone Alert Days' could increase by 68% due to projected regional warming alone. NMED-Patz Exhibit 4. But according to the IPCC, the projected increase in stagnant air masses for the Midwest and Northeast may exacerbate this problem even more. Ozone is especially dangerous to children with asthma. Recall the findings during the 1996 Atlanta Olympics when traffic restrictions resulted in a 28% decrease in ground-level ozone, and a subsequent 42% decline in asthma admissions to emergency rooms.

Pollen, another air contaminant, may increase with elevated temperature and CO_2. For instance, studies have found that a doubling of the atmospheric CO_2 concentration stimulates ragweed-pollen production by over 50%. NMED-Patz Exhibit 5.

Many infectious diseases are sensitive to climate fluctuations. For example, 67% of reported water-borne disease outbreaks in the U.S. between 1948-1994 were preceded by very heavy rainfall; projections are for increases in extreme rainfall and runoff, placing more risk on already deteriorating water systems in many cities. Combined

sewage overflows (CSOs) will likely become a more frequent problem. West Nile virus (WNV) emerged for the first time in North America during the record hot July, 1999. While international transport likely explained its entry in the U.S., this particular strain of WNV requires warmer temperatures than other strains around the globe. Elevated WNV transmissions during the epidemic summers of 2002-2004 in the U.S. were linked to above-average temperatures. Please refer to the New Mexico Climate Change Impacts Report for more specific information regarding infectious diseases in the Southwest, such as hantavirus and coccidioidomycosis, or Valley Fever. NMED-Norton Exhibit 2. I also would refer the Board to the health reports prepared by the State of California, which contain substantial information about the potential health effects of climate change. NMED-Patz Exhibits 6 and 7.

As noted earlier, heat itself has an impact on public health. According to a study analyzing NOAA weather data for trends in heat index and heat wave frequency, heat stress in American cities has increased dramatically over the past 50 years. For each of 113 weather stations with complete data records, the trend in the frequency of extreme heat stress between 1948 and 1995 was computed. Both single-day events and multi-day heatwaves were considered. The trend was upward over most of the U.S. The trend in nighttime extremes was generally larger than for the daytime, consistent with the trends in mean conditions. The data also revealed an expected pattern in which summertime heat is more severe in the southeast, south-central, and southwest regions of the U.S. For a more detailed discussion of health impacts, please refer to EPA's Synthesis Assessment Product (SAP 4.6), EPA's Endangerment Findings report, and the North American chapter of the 4th Assessment report of IPCC. NMED-Patz Exhibits 8, 9, and 10.

B. CAN WE ADAPT TO CLIMATE CHANGE RISKS?

Relying on adaptation alone is a dangerous strategy. Building adaptive capacity takes time and is unlikely to be reliable for climatic changes that might be more rapid or more extreme than expected. In addition, a majority of greenhouse gas emissions in the future will come from developing countries. Therefore, by relying on adaptation to deal with climate change, the U.S. provides no basis for leadership or persuasion to enlist developing countries to reduce their emissions: in the end, we may have to adapt even more. Global greenhouse gas emissions have been accelerating over the past decade and outside the upper end of scenarios predicted a decade ago.

C. ARE THERE PUBLIC HEALTH CO-BENEFITS FROM REDUCTING GREENHOUSE GAS EMISSIONS?

Considering the multiple health outcomes and the potential for adverse synergies between global warming, urban sprawl, and land degradation, climate change poses a major threat to the health of the U.S. population. The policy changes needed to address this problem are going to be very large if we are serious about protecting the public from the adverse health effects of climate change. Adopting a modest emissions reductions policy, which may be riddled with loopholes, in the interest of pushing the U.S. to finally adopt a climate policy seems a like a risky approach. With such large ramifications at stake and so many potential health co-benefits to be gained by reducing greenhouse gas emissions, major policy measures to mitigate climate change seem like an obvious component to protecting our health.

Scientific assessments caution that climate change will have dangerous synergies with other environmental public health risks, and so must not be viewed as an isolated

health risk. Dangerous synergies will include, for example, the 'urban heat island' effect over sprawling cities with asphalt highways; destruction of storm-buffering coastal wetlands, e.g, near New Orleans; and increased allergens in the air along with a lengthening ozone pollution season.

Yet these dangerous synergies also point to potential co-benefits of mitigating greenhouse warming. There are potentially large opportunities and co-benefits in addressing the health risks of global warming. Certainly, our public health infrastructure must be strengthened, including fortified water supply systems, heat and storm early warning and response programs, and enhanced disease modeling and surveillance. However, energy policy now becomes one and the same as public health policy. Reducing fossil fuel burning will: (1) reduce air pollution; thel reduction of fossil fuel burning reduces NOx and CO emissions, as well as SO2, PM2.5, Hg, VOC and/or air toxic emissions (depending on the sectors, fuels, and technologies affected); (2) improve our fitness; only 40% of the U.S. population meets the minimum daily recommended level of exercise and 60% of Americans are overweight, and these percentages would improve if urban transportation planning caused more Americans to travel by foot or bike and public transportation rather than by car; and (3) lessen potential greenhouse gas emissions and subsequent global warming. Most of the ten leading causes of death in the US are linked either to sedentary lifestyles, air pollution, or motor vehicle crashes.

In short, the challenges posed by climate change urgently demand reducing greenhouse gases, improving public health infrastructure *and* implementing energy conservation/urban planning policies. As such, climate change can present both enormous

health risks and opportunities quite directly via improved fitness, reduced obesity (with its multitude of associated diseases), and improved air quality.

The scientific rationale for regulating CO_2 is absolutely clear when considering these health risks. Fragmented strategies and cost/benefit analysis undermine our ability to address these health risks. My colleague and energy policy expert, Dr. Greg Nemet, shared with me his concern that CO_2 regulation based on a cost/benefit risk assessment analysis only weakly captures these health risks because: (1) most impacts of U.S. emissions will be felt outside the U.S.; (2) impact assessments focus on likely ranges, and ignore tails (or extremes) of distributions; and (3) impacts will be mostly in the future, and so will be discounted heavily. Thus, a worrisome outcome is that EPA could end up regulating CO_2, but set only modest reduction targets which do not adequately protect the health of Americans. From my standpoint as a public health scientist, I view the health threats of climate change as extremely large in magnitude, and therefore requiring equivalently significant policy change – both in areas of public health preparedness and in greenhouse gas mitigation.

Dr. Tracey Holloway, a climate-air pollution expert at SAGE, pointed out to me that policy analyses for Europe have quantified the economic and physical interactions between climate change and air quality, finding that integrated policies to address both issues simultaneously could reduce total costs by well over 1 billion Euro/yr by 2020 versus the cost of considering air quality and climate separately.

II. CONCLUSIONS AND RECOMMENDATIONS

In summary, many human health outcomes are strongly influenced by climate; as climate changes, so to will many of these climate-sensitive diseases. While independently there are many different preventive measures to reduce each risk, the broadest prevention can be achieved by moving upstream in the causal pathway of diseases. Therefore, stemming the tide of greenhouse gas emissions (the root cause of climate change) should be considered as a key component of public health interventions to protect the public from climate change.

These broad and interconnected exposures require a well-coordinated, cross-sector and comprehensive disease prevention strategy. In addition to enhancing disease preparedness, this strategy includes proactive energy conservation and transportation policies, which concurrently provide substantial health co-benefits.

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REVIEWS

Impact of regional climate change on human health

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The World Health Organisation estimates that the warming and precipitation trends due to anthropogenic climate change of the past 30 years already claim over 150,000 lives annually. Many prevalent human diseases are linked to climate fluctuations, from cardiovascular mortality and respiratory illnesses due to heatwaves, to altered transmission of infectious diseases and malnutrition from crop failures. Uncertainty remains in attributing the expansion or resurgence of diseases to climate change, owing to lack of long-term, high-quality data sets as well as the large influence of socio-economic factors and changes in immunity and drug resistance. Here we review the growing evidence that climate–health relationships pose increasing health risks under future projections of climate change and that the warming trend over recent decades has already contributed to increased morbidity and mortality in many regions of the world. Potentially vulnerable regions include the temperate latitudes, which are projected to warm disproportionately, the regions around the Pacific and Indian oceans that are currently subjected to large rainfall variability due to the El Niño/Southern Oscillation sub-Saharan Africa and sprawling cities where the urban heat island effect could intensify extreme climatic events.

Global average temperatures are projected to increase between 1.4 and 5.8 °C by the end of this century[1]; an associated rise in sea level is also expected. The number of people at risk from flooding by coastal storm surges is projected to increase from the current 75 million to 200 million in a scenario of mid-range climate changes, in which a rise in the sea level of 40 cm is envisaged by the 2080s (ref. 2). Extremes of the hydrologic cycle (such as floods and droughts) are projected to increase with warmer ambient temperatures. Evidence is mounting that such changes in the broad-scale climate system may already be affecting human health, including mortality and morbidity from extreme heat, cold, drought or storms; changes in air and water quality; and changes in the ecology of infectious diseases[3–5].

We reviewed both empirical studies of past observations of climate-health relationships, and model simulation studies of projected health risks and regional vulnerability associated with future climate change. Here we focus on the health implications of climate variability, past and present climate change impacts on human health, future projections and uncertainties. This review primarily examines relatively direct-acting temperature effects, while recognizing that other major risk pathways exist, for instance, altered storm patterns, hydrologic extremes, and sea-level rise.

Health implications of climate variability

Non-infectious health effects. The summer of 2003 was probably Europe's hottest summer in over 500 years, with average temperatures 3.5 °C above normal[6–8]. With approximately 22,000 to 45,000 heat-related deaths occurring across Europe over two weeks in August 2003 (refs 9 and 10), this is the most striking recent example of health risks directly resulting from temperature change. Judging from this extreme event, changes in climate variability associated with long-term climate change could be at least as important for future risk assessment as upward trends in mean temperature.

The European heatwave in 2003 was well outside the range of expected climate variability[8]. In addition, comparisons of climate model outputs with and without anthropogenic drivers show that the risk of a heatwave of that magnitude had more than doubled by 2003 as a result of human-induced climate change[3]. The demonstration of a causal link between global warming and the occurrence of regional heatwaves indicates a potential for more frequent and/or more severe heatwaves in a future warmer world.

On local and regional scales, changes in land cover can sometimes exacerbate the effect of greenhouse-gas-induced warming, or even exert the largest impact on climatic conditions. For example, urban 'heat islands' result from lowered evaporative cooling, increased heat storage and sensible heat flux caused by the lowered vegetation cover, increased impervious cover and complex surfaces of the cityscape. Dark surfaces such as asphalt roads or rooftops can reach temperatures 30–40 °C higher than surrounding air[11]. Most cities show a large heat island effect, registering 5–11 °C warmer than surrounding rural areas[12]. But the effects of land cover change on climate are not limited to small areas: at the scale of the entire continental USA, Kalnay and Cai[13] estimated that land-cover changes (from both agriculture and urban areas) caused a surface warming of ~0.27 °C per century. Also, in southeast China, a warming of ~0.05 °C per decade since 1978 has been attributed to land-use change from urban sprawl[14].

Exposure to both extreme hot and cold weather is associated with increased morbidity and mortality, compared to an intermediate 'comfortable' temperature range[15]. Heat mortality follows a J-shaped function with a steeper slope at higher temperatures[16]. The comfortable or safest temperature range is closely related to mean temperature, with an upper bound from as low as 16.5 °C for the Netherlands and 19 °C for London[17], to as high as 29 °C in Taiwan[18]. Hot days occurring earlier in the summer season have a larger effect than those occurring later[17]. It should be noted that although the majority of temperature–mortality studies have taken place in developed

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countries and in regions with temperate climates, the same pattern of temperature–mortality relationship found in European and North American cities occurs in São Paulo, Brazil, a developing city with subtropical conditions[19].

In summary, although most studies to date show clear vulnerability to heat in cooler temperate regions, tropical regions may well show a similar sensitivity as location-specific temperatures rise.

Climatic influences on regional famines are another well-recognized climate–health association. Malnutrition remains one of the largest health crises worldwide and according to the WHO, approximately 800 million people are currently undernourished, with close to half of these living in Africa[20]. Droughts and other climate extremes have direct impacts on food crops, and can also influence food supply indirectly by altering the ecology of plant pathogens. Projections of the effect of climate change on food crop yield production globally appear to be broadly neutral, but climate change will probably exacerbate regional food supply inequalities[21].

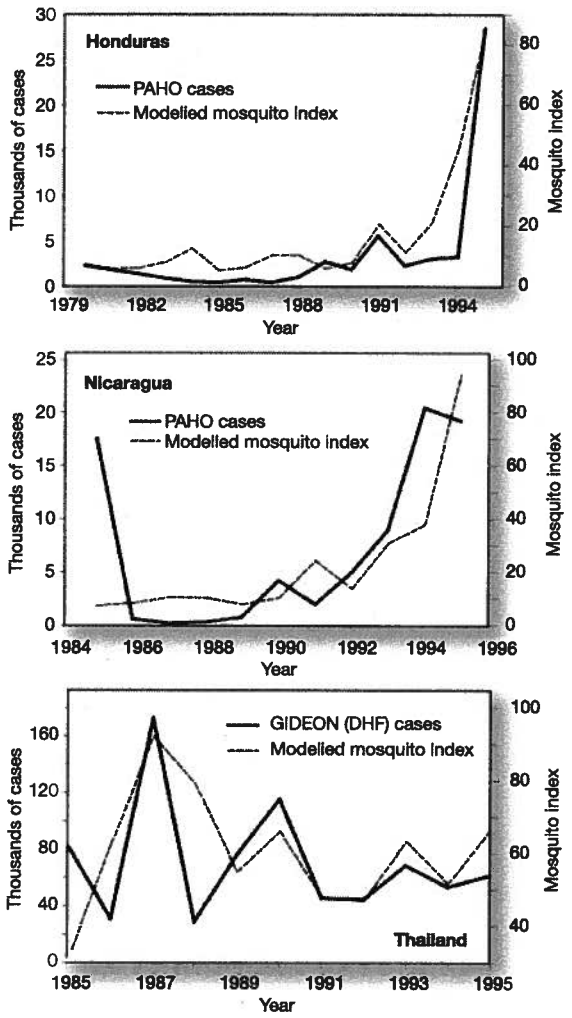

Figure 1 | Correlation between simulated, climate-driven variations in *Aedes aegypti* mosquito density and observed variations in dengue and DHF cases. Using a computer model of mosquito physiology and development, estimated changes in the relative abundance of *Aedes aegypti* that were driven only by month-to-month and year-to-year variations in temperature, humidity, solar radiation and rainfall were analysed. The simulated, climate-induced variations in mosquito density were then compared to reported cases of dengue and DHF across many nations of the world that covered at least one degree of latitude and longitude and had at least five years of dengue caseload data. In many countries of Central America and Southeast Asia, the relationship is statistically significant ($P < 0.05$). For example, climate-driven fluctuations in *Ae. aegypti* densities appear to be related to annual variations in dengue/DHF cases in Honduras, Nicaragua and Thailand as shown. These represent relatively small-area countries; for larger countries endemic for dengue such as Brazil, China, India and Mexico, the association is not significant, as might be expected because the disease data was at the country level. Graphs adapted from ref. 32.

Infectious diseases. Climatic variations and extreme weather events have profound impacts on infectious disease. Infectious agents (such as protozoa, bacteria and viruses) and their associated vector organisms (such as mosquitoes, ticks and sandflies) are devoid of thermostatic mechanisms, and reproduction and survival rates are thus strongly affected by fluctuations in temperature[4,22]. Temperature dependencies are seen in correlations between disease rates and weather variations over weeks, months or years[23] and in close geographic associations between key climate variables and the distributions of important vector-borne diseases[24,25].

Malaria transmission has been associated with anomalies of maximum temperature in the highlands of Kenya[26]. Several studies of long-term trends in malaria incidence and climate in Africa, however, have not found a link to temperature trends, emphasizing instead the importance of including other key determinants of malaria risk such as drug resistance, human migration and immune status, inconsistent vector- or disease-control programmes, and local land-use changes[27–30]. However, in the highland Debre Zeit sector of central Ethiopia an association has been documented between increasing malaria prevalence and incidence with concomitant warming trends from 1968 to 1993 (ref. 31). Controlling for confounding factors, the association could not be explained by drug resistance, population migration, or level of vector-control efforts. In short, studies of the association of malaria and past climate in the African Highlands remains controversial in part due to varying quality of long-term disease data across sites in Africa, and in part due to the difficulty in adequately controlling for demographic and biological (drug resistance) data. A definitive role of long-term climate trends has not been ascertained.

Dengue fever and the more serious form of this disease, dengue haemorrhagic fever (DHF), are caused by the world's most prevalent mosquito-borne virus. All strains of the dengue virus are carried principally by the *Aedes aegypti* mosquito. This mosquito is strongly affected by ecological and human drivers, particularly the density of water-bearing containers, but is also influenced by climate, including variability in temperature, moisture and solar radiation. For relatively small countries with presumably some climate uniformity, a climate-based dengue model has been developed that strongly correlates with the inter-annual variability in dengue cases reported at the national level (Fig. 1)[32].

A few examples of other vector-borne diseases demonstrating variance with climate include the Ross River virus in Australia[33,34], and plague[35] in the American southwest. Bluetongue, a disease of livestock, has increased its northern range in Europe since 1998, paralleling trends in warming and controlling for many biological and socioeconomic factors[36].

Temperature has also been found to affect food-borne infectious diseases. For example, higher than average temperatures contribute to an estimated 30% of reported cases of salmonellosis across much of continental Europe[37]. In the UK, the monthly incidence of food poisoning is most strongly associated with the temperatures occurring in the previous two to five weeks[38].

El Niño/Southern Oscillation and infectious diseases. With the exception of seasonal variability, the El Niño/Southern Oscillation (ENSO) is the strongest naturally occurring source of climate variability around the globe[39]. Studies of malaria have revealed the health impacts of interannual climate variability associated with El Niño, including large epidemics on the Indian subcontinent[40], in Colombia[41], Venezuela[42] and Uganda[43]. Rift Valley fever epidemics

between 1950 and 1998 have coincided with unusually high rainfall in East Africa associated with ENSO-related Pacific and Indian Ocean sea surface temperature (SST) anomalies[44]. While more than three quarters of the Rift Valley Fever outbreaks between 1950 and 1988 occurred during warm ENSO event periods[45], some epidemics have also occurred in years with no El Niño, and the model has not been validated against new epidemics.

A 'wavelet analysis' method was recently used to incorporate host immunity and pathogen population dynamics of DHF in Thailand. A spatial-temporal travelling wave explained a three-year period cycle in disease incidence, starting in Bangkok, moving radially at a speed of 148 km per month[46]. In a subsequent study that controlled for this intrinsic synchronization, El Niño remained as a significant determinant of dengue epidemics that cycled every two to three years from 1986 to 1992 in Thailand[47].

Hantavirus pulmonary syndrome in the American southwest can be predicted on the basis of ENSO events; following the 1991–92 El Niño, associated heavy rainfall led to an increase in the

Table 1 | Global burden of climate-change-attributable disease

Region	CVD		Diarrhoea			Malaria			Floods			
	Mortality*	Risk‡	Mortality*	Disease†	Risk‡	Mortality*	Disease†	Risk‡	Mortality*	Disease†	Risk‡	
											Inland	Coastal
AFR-D	1	1.007	5	154	1.08	5	178	1.02	0	1	1.36	1.64
AFR-E	1	1.005	8	260	1.08	18	682	1.14	0	3	1.48	1.18
AMR-A	0	1	0	0	1	0	0	1.51	0	4	4.93	1.19
AMR-B	1	1.004	0	0	1	0	3	1.15	1	67	2.13	2.27
AMR-D	0	1.005	1	17	1.02	0	0	1.08	0	5	1.78	4.64
EMR-B	0	1.003	0	14	1	0	0	1	0	6	2.67	1.75
EMR-D	1	1.003	8	277	1.09	3	112	1.29	1	46	3.05	3.91
EUR-A	0	0.999	0	0	1	0	0	1	0	3	3.55	1.14
EUR-B	0	0.999	0	6	1.01	0	0	1	0	4	1.82	6.31
EUR-C	0	0.998	0	3	1	0	0	1.48	0	1	2.35	1.04
SEAR-B	1	1.007	1	28	1	0	0	1	0	6	1.79	1.39
SEAR-D	7	1.007	22	612	1.09	0	0	1.01	0	8	1.12	1.04
WPR-A	0	0.999	0	0	1	0	0	1.48	0	1	1.76	1.04
WPR-B	0	1	2	89	1.01	1	43	1.42	0	37	1.62	1.05
World	12§	-	47	1,459	-	27	1,018	-	2	193	-	-

Region	Malnutrition			All causes		Total deaths per million	Total DALYs per million
	Mortality*	Disease†	Risk‡	Mortality*	Disease†	Mortality*	Disease†
AFR-D	8	293	1.02	19	626	66.83	2,185.78
AFR-E	9	323	1.02	36	1,267	109.4	3,839.58
AMR-A	0	0	1	0	4	0.15	11.85
AMR-B	0	0	1	2	71	3.74	166.62
AMR-D	0	0	1	1	23	10.28	324.15
EMR-B	0	0	1	1	20	5.65	147.57
EMR-D	9	313	1.08	21	748	61.3	2,145.91
EUR-A	0	0	1	0	3	0.07	6.66
EUR-B	0	0	1	0	10	1.04	48.13
EUR-C	0	0	1	0	4	0.29	14.93
SEAR-B	0	0	1	2	34	7.91	117.19
SEAR-D	52	1,918	1.17	80	2,538	65.79	2,080.84
WPR-A	0	0	1	0	1	0.09	8.69
WPR-B	0	0	0.99	3	169	2.16	111.36
World	77	2,846	-	166	5,517	27.82	925.35

*Estimated mortality in thousands attributable to climate change in 2000 (compared to baseline climate of 1961–1990).
†Estimated disease burden in thousands of DALYs attributable to climate change in 2000.
‡Projected changes in relative risk for 2030.
§Heat-related deaths without subtracting potential reductions in cold-related deaths; this value was therefore not included in the aggregate estimates of mortality due to climate change.
The data in Table 1 are taken from ref. 57. The region key is taken from ref. 57. AFR-D: Algeria, Angola, Benin, Burkina Faso, Cameroon, Cape Verde, Chad, Comoros, Equatorial Guinea, Gabon, Gambia, Ghana, Guinea, Guines-Bissau, Liberia, Madagascar, Mali, Mauritania, Mauritius, Niger, Nigeria, Sao Tome and Principe, Senegal, Seychelles, Sierra Leone, Togo.
AFR-E: Botswana, Burundi, Central African Republic, Congo, Côte d'Ivoire, Democratic Republic of the Congo, Eritrea, Ethiopia, Kenya, Lesotho, Malawi, Mozambique, Namibia, Rwanda, South Africa, Swaziland, Uganda, United Republic of Tanzania, Zambia, Zimbabwe.
AMR-A: Cuba, Canada, United States of America.
AMR-B: Antigua and Barbuda, Argentina, Bahamas, Barbados, Belize, Brazil, Chile, Colombia, Costa Rica, Dominica, Dominican Republic, El Salvador, Grenada, Guyana, Honduras, Jamaica, Mexico, Panama, Paraguay, Saint Kitts and Nevis, Saint Lucia, Saint Vincent and the Grenadines, Suriname, Trinidad and Tobago, Uruguay, Venezuela.
AMR-D: Bolivia, Ecuador, Guatemala, Haiti, Nicaragua, Peru.
EMR-B: Bahrain, Cyprus, Iran, Jordan, Kuwait, Lebanon, Libyan Arab Jamahiriya, Oman, Qatar, Saudi Arabia, Syrian Arab Republic, Tunisia, United Arab Emirates.
EMR-D: Afghanistan, Djibouti, Egypt, Iraq, Morocco, Pakistan, Somalia, Sudan, Yemen.
EUR-A: Andorra, Austria, Belgium, Croatia, Czech Republic, Denmark, Finland, France, Germany, Greece, Iceland, Ireland, Israel, Italy, Luxembourg, Malta, Monaco, the Netherlands, Norway, Portugal, San Marino, Slovenia, Spain, Sweden, Switzerland, United Kingdom.
EUR-B: Albania, Armenia, Azerbaijan, Bosnia and Herzegovina, Bulgaria, Georgia, Kyrgystan, Poland, Romania, Slovakia, Tajikistan, Macedonia, Turkey, Turkmenistan, Uzbekistan, Yugoslavia.
EUR-C: Belarus, Estonia, Hungary, Kazakhstan, Latvia, Lithuania, Moldova, Russian Federation, Ukraine.
SEAR-B: Indonesia, Sri Lanka, Thailand.
SEAR-D: Bangladesh, Bhutan (Democratic People's Republic of), Korea, India, Maldives, Myanmar, Nepal.
WPR-A: Australia, Brunei, Darussalam, Japan, New Zealand, Singapore.
WPR-B: Cambodia, China, Cook Islands, Fiji, Kiribati, Lao, Malaysia, Marshall Islands, Micronesia, Mongolia, Nauru, Niue, Palau, Papua New Guinea, Philippines, Republic of Korea, Samoa, Solomon Islands, Tonga, Tuvalu, Vanuatu, Vietnam.

rodent populations that preceded human cases of disease[48]. Based on these climate/ecology/disease relationships, a climate- and GIS (Geographic Information System)-based model was developed that predicted disease risk reasonably well for the following strong El Niño event of 1997–98 (ref. 49).

Waterborne diseases, such as childhood diarrhoeal disease, are also influenced by El Niño, as was observed with the 1997–98 El Niño event in Peru. During that unseasonable winter, the ambient temperature in Lima increased more than 5 °C above normal, and the number of daily admissions for diarrhoea increased by more than twofold, compared to expected trends[50].

Cholera has varied with climatic fluctuations and SSTs affected by the ENSO phenomenon over multi-decadal time periods in Bangladesh[51]. In the Bay of Bengal, upward trends in cholera also have been linked to longer-term climate changes (that is, changes over approximately a century), with weak cholera/ENSO links found during 1893–1940, and strong and consistent associations occurring during the more pronounced ENSO fluctuations between 1980–2001 (ref. 52). One ecologically based hypothesis for this link involves copepods (zooplankton), which feed on algae, and can serve as reservoirs for *Vibrio cholerae* and other enteric pathogens[53]; copepods bloom in response to the warming SSTs generally associated with El Niño.

Understanding interannual cycles of cholera and other infectious diseases (as seen above for dengue fever), however, requires the combined analyses of both environmental exposures and intrinsic host immunity to a disease. When these factors are considered together, interannual variability of cholera is strongly correlated to SSTs in the Bay of Bengal, ENSO, the extent of flooding in Bangladesh across short time periods (<7 years), and to monsoon rains and Brahmaputra river discharge for longer period climate patterns (>7 years)[54].

Although it is not clear whether and how ENSO dynamics will change in a warmer world, regions that are currently strongly affected by ENSO (for example, southeast Asia, southern and east Africa, the southwest USA, and various regions of South America) could experience heightened risks if ENSO variability, or the strength of El Niño events intensifies.

Land use, local climate and infectious disease. Just as the 'urban heat island' (mentioned above) exacerbates heatwaves, so too can land use change influence transmission of infectious diseases. Land cover may affect mosquito habitat by changing local temperature and humidity. For example, temperatures were significantly higher in communities in highland Uganda bordering cultivated swamps compared with natural ones[55], and average minimum temperatures were associated with the number of *Anopheles gambiae sensu lato* (*s.l.*) mosquitoes per house after adjustment for potential confounding variables. Of course, in some locations, rainfall is more important than temperatures or land use, and in some locations malaria epidemics in the highlands of Africa are more influenced by changing disease-control efforts than any other factors[29].

Past and present climate-change impacts

In the most comprehensive, peer-reviewed and quantitative climate–health assessment to date, the World Health Organization (WHO) examined the global burden of disease already attributable to anthropogenic climate change up to the year 2000 (ref. 20); WHO also made model-based forecasts of the health risks from global climate change until 2030 (refs 56, 57).

The study made generally conservative assumptions about climate–health relationships (for example, that socioeconomic conditions would prevent a climate-driven spread of vector-borne disease from endemic tropical regions to temperate regions), and health impacts were included only if quantitative models were available. An assessment over such a broad range of health impacts is by nature approximate, as there are significant uncertainties in all climate change–disease models. The study indicates that the climatic changes that have occurred since the mid-1970s could already be causing over 150,000 deaths and approximately five million 'disability-adjusted life years' (DALYs) per year through increasing incidences of diseases such as diarrhoea (temperature effects only), malaria and malnutrition that occur mainly in developing countries[57] (Table 1, Fig. 2).

The WHO assessment emphasized that actions to adapt to a changing climate will require regional assessments of vulnerability to specific health risks, and interventions that are geographically and temporally targeted on highly susceptible populations.

Future projections and uncertainties

The WHO extended its estimates of morbidity and mortality caused by human-induced climate change to the year 2030, following

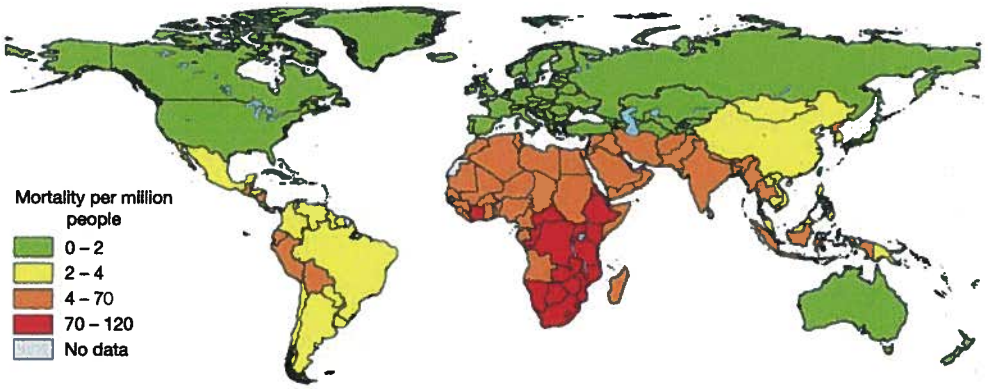

Figure 2 | WHO estimated mortality (per million people) attributable to climate change by the year 2000. The IPCC 'business as usual' greenhouse gas emissions scenario, 'IS92a' and the HadCM2 GCM of the UK Hadley Centre were used to estimate climate changes relative to 'baseline' 1961–1990 levels of greenhouse gases and associated climate conditions. Existing quantitative studies of climate–health relationships were used to estimate relative changes in a range of climate-sensitive health outcomes including: cardiovascular diseases, diarrhoea, malaria, inland and coastal flooding, and malnutrition, for the years 2000 to 2030. This is only a partial list of potential health outcomes, and there are significant uncertainties in all of the underlying models. These estimates should therefore be considered as a conservative, approximate, estimate of the health burden of climate change. Even so, the total mortality due to anthropogenic climate change by 2000 is estimated to be at least 150,000 people per year. Details on the methodology are contained in ref. 57.

Hadley Centre global climate model (GCM) projections for a range of greenhouse-gas emissions scenarios[58]. It estimates that the climate-change-induced excess risk of the various health outcomes will more than double by the year 2030 (ref. 57). Large increases are predicted for the relative risk of flooding and more modest changes in diseases such as malaria, malnutrition and diarrhoea (Table 1). However, it is important to note that these small relative changes may actually cause far greater aggregate disease burdens. In sub-Saharan Africa, for example, flooding currently kills less than one person per million annually, while malaria kills over 1,600 per million and diarrhoea kills over 1,000 per million (ref. 57).

To consider changes in future heatwave probabilities, GCM projections of future climate for conditions contributing to heatwaves are now capable of estimating the occurrence of stagnant, warm air masses that can determine the severity of a heatwave, including variables such as consecutive nights (three or more) with high minimum night-time temperatures[59]. A recent analysis of the 1995 Chicago and 2003 Europe heatwaves predicted intensified magnitude and duration of heatwaves over portions of Europe and the United States, suggesting that heatwaves in Chicago and Paris will be 25% and 31% more frequent, respectively, by 2090 and that the average length of a heatwave in Paris will have increased from 8–13 days to 11–17 days. Large increases in heatwaves were also projected for the western and southern USA and the Mediterranean region[59].

Data from the MARA (Mapping Malaria Risk in Africa) project have been applied to global climate projections to examine potential changes in malaria risk over regions of Africa[60]. Excluding any increase in population, an increase of 16–28% in person-month exposure (number of people exposed per month) to malaria risk by year 2100 was determined[60]. However, like all previous continental or global models of malaria–climate relationships, the study fails to account for non-climatic determinants or the variation of specific climate–disease relationships among locations[61,62].

Extrapolation from statistically based models into the future is of limited value. There may be evidence that malaria is increasing in the highlands of Africa owing to climate change, but methods used to detect it are still controversial and do not convincingly prove or disprove the association. To assess the health risks of mid- to long-term future climate projections, a concerted effort combining the use of process-based models (capturing the biology of the malaria system) alongside statistical modelling will be needed.

Regional assessments of health impacts. Climate-change projections from GCMs, such as a recent set of simulations performed in preparation for the Intergovernmental Panel on Climate Change (IPCC) Fourth Assessment Report[63], are increasing in resolution, but are still not appropriate for analysing disease patterns at scales smaller than a few grid boxes of area 250 km^2. For example, local patterns of climate that are strongly influenced by subtle changes in topography (slope and aspect, rainshadow effects, orographic precipitation), large water bodies, coastlines and other geographic features may be important determinants of disease ecology.

Moving from large-scale climate projections to smaller spatial scales requires the application of 'downscaling' techniques that bring additional information to bear on the region in question. Downscaling methods fall into two broad categories: dynamical downscaling, using high-resolution, regional climate models[64,65], and statistical downscaling, based on statistical relationships between large-scale predictor variables and regional predictants (Table 2).

A dynamical downscaling study with the aim of determining the impact of potential climate changes over the next 50 years on air pollution in the eastern USA[66] reported that under the high-emission 'A2' IPCC scenarios, daily average ozone levels increase by 3.7 p.p.b. across the eastern USA, with the most polluted cities today experiencing the greatest increase in temperature-related ozone pollution (Fig. 3). Across 15 selected cities in this region, the average number of days exceeding the 8-hour ozone standard increased by 60%—from 12 to almost 20 days per summer by the 2050s (ref. 67). Assuming constant population and dose–response characteristics, an independent dynamical downscaling study[64] (refs 64 and 67 both stem from the modeling work of ref. 66) projected that ozone-related deaths from climate change will increase by ~4.5% for the mid-2050s (using the 'A2' emissions scenario), compared with the levels of the 1990s. Considering the potential population exposed to outdoor air pollution (in the millions), this seemingly small relative risk actually translates to quite a substantial attributable health risk. There is significant uncertainty associated with these findings, as they are based on a single emissions scenario, one GCM simulation, and many assumptions about regional ozone precursor emissions.

Table 2 | Differences between dynamical and statistical downscaling

	Benefits	Drawbacks	Applications
Dynamical downscaling	• Simulates climate mechanisms • No *a priori* assumptions about how current and future climate are related • 'State of the science' tools • Continually advancing computers are making RCMs faster and cheaper to run • Encourages collaborations between health and climate scientists	• Expensive, in terms of computer resources and professional expertise • Results may be sensitive to uncertain parameterisations • Biases in the GCM (providing boundary conditions) may propagate to regional scale • Output from models may not be in a format well-suited to health analysis—additional data processing often required	• Health responses associated with climate extremes and nonlinear variability • Data-poor areas • Connecting outcomes with climate processes • Include land-use impacts on climate or health outcomes
Statistical downscaling (especially regression methods)	• Much cheaper (runs quickly on desktop computers with free software) • Builds on the statistical expertise common among public health researchers • May correct for biases in GCM • Allows for the assessment of climate results over a range of GCMs and emission scenarios	• Assumes relationships between local and large-scale climate remain constant • Does not capture climate mechanisms • Not well suited to capturing variance or extreme events	• Climate means, and variability with some limitations • Data-rich regions, especially Northern Hemisphere mid-latitudes • Compare present with projected climate in a consistent framework • Test a range of inputs • Variable scales, down to individual measurement sites

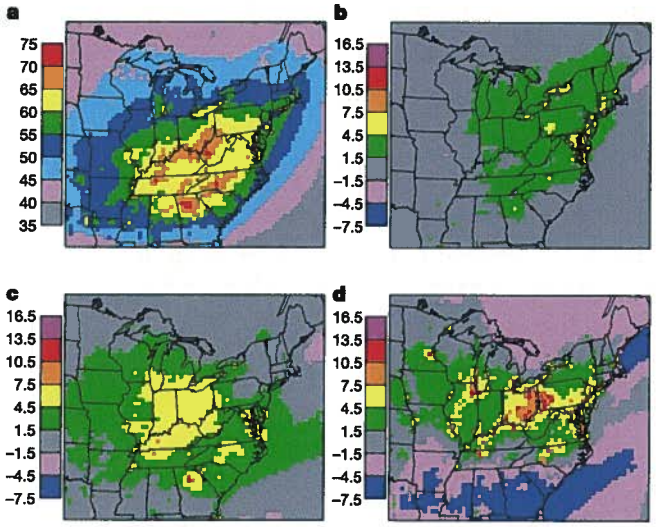

Figure 3 | Simulated ozone air pollution over the eastern United States by using a downscaled climate model linked to a regional air pollution model. a, Baseline summertime average daily maximum 8-hour O_3 concentrations for the 1990s. **b–d**, The following panels show changes in summertime-average daily maximum 8-hour O_3 concentrations for the 2020s (**b**), the 2050s (**c**), and the 2080s (**d**) over the region based on IPCC A2 scenario simulations relative to the 1990s, in parts per billion. Five consecutive summer seasons were simulated in each decade starting with the NASA Goddard Institute for Space Studies (GISS) Atmosphere-Ocean Global Climate Model, with results subsequently downscaled using the mesoscale regional climate model (MM5), and finally coupled to the Community Multiscale Air Quality (CMAQ) model. Simulation results for the 2020s, 2050s and 2080s indicate that summertime average daily maximum 8-hour O_3 concentrations increase by 2.7, 4.2 and 5.0 p.p.b., respectively, as a result of regional climate change. The data were taken from ref. 66.

Statistical downscaling is most useful for health assessment in data-rich regions. On the basis of statistical downscaling techniques, heat-related deaths in California are estimated to more than double by the year 2100 (ref. 68).

Pursuing early warning systems. Current early warning systems for infectious diseases are beginning to show some utility. For example, over two-thirds of the inter-annual variability of malaria in Botswana can be predicted from the SSTs and associated monthly rainfall[69]. For more direct health impacts from heatwaves, although uncertainty still remains as to which weather parameters are most hazardous, a number of studies have consistently identified high minimum (night-time) temperatures, duration, and early seasonal occurrence of heatwaves as particularly dangerous conditions[70]. Therefore, early warning systems may offer some health protection from the effects of heatwaves. For example, after the 1995 heatwave in the United States, the city of Milwaukee initiated an 'extreme heat conditions plan' involving local agencies, communications tests, stepped responses to early forecasts, a 24-hour 'hotline' and other interventions. Reductions in heat-related morbidity (measured by emergency ambulance runs) and mortality were reduced by 49% from expected levels during a heatwave in 1999, and were not attributable to differences in heat levels alone[71].

Currently, over two dozen cities worldwide have a 'synoptic-based' weather-watch warning system, which focuses monitoring on dangerous air masses[72]. These systems successfully forecast most days with excess deaths in Rome during the 2003 heatwave[73], and have been implemented successfully in Shanghai, for example[74]. However, variability in predictability between cities suggests that systems must be location-specific, requiring the input of considerable amounts of health-related and meteorological data for each locale[75].

Health and regional climate change

Two main climatic impacts on health at a regional scale emerge from this review: direct heat-related mortality and morbidity, and a climate-mediated change in the incidence of infectious diseases.

Heat-related mortality is dominated by the difference between temperature extremes and the mean climate—especially early in summer when people have not yet become accustomed to higher temperatures—rather than by gradual increases in mean temperatures. Projections of future climate suggest such increases in extremes in relation to mean temperatures may occur particularly in the mid-latitudes. In addition, the effect of heatwaves is exacerbated in large cities owing to the urban heat island effect. As urban areas and urban population grow, vulnerability to heat-related mortality seems likely to increase in the future.

Studies of climatic influences on infectious diseases have mainly focused on the influence of ENSO. ENSO has been found to be related to incidences of malaria in South America, rift valley fever in east Africa, dengue fever in Thailand, hantavirus pulmonary syndrome in the southwestern USA, childhood diarrhoeal disease in Peru and cholera in Bangladesh. It is unclear at this stage whether global warming will significantly increase the amplitude of ENSO variability, but if so, the regions surrounding the Pacific and Indian oceans are expected to be most vulnerable to the associated changes in health risks.

Potential impacts of long-term trends in mean temperatures on health, for example, on malaria incidence in the African highlands, have not been reliably detected. The data available at present do not allow robust control for non-climatic confounding factors such as socio-economic influences, immunity patterns and drug resistance effects. However, regions bordering areas with high endemicity of climate-sensitive diseases, where temperatures at present limit the geographic distribution of disease (such as malaria in the African highlands) could be at risk in a warmer climate.

Early warning systems both for heatwaves and for expected outbreaks of infectious diseases can help to adapt to some of the effects of a changing climate, through measures such as opening air-conditioned shopping malls at night-time to those who are most vulnerable to heat, or providing prophylactic treatment to those in danger from infectious diseases. However, population vulnerability still greatly depends on economic and other determinants of a society's capacity to provide such measures.

Land use and land cover change, as mentioned above, can magnify the effects of extreme climatic events, both on direct health outcomes (for example, heat mortality), and on ecologically mediated infectious diseases in any region of the world. Therefore, to assess accurately future climate-change impacts on health, future projections of land-use change must be considered as well.

As illustrated in Fig. 2, the regions with the greatest burden of climate-sensitive diseases are also the regions with the lowest capacity to adapt to the new risks. Africa—the continent where an estimated 90% of malaria occurs—has some of the lowest per capita emissions of the greenhouse gases that cause global warming. In this sense, global climate change not only presents new region-specific health risks, but also a global ethical challenge. To meet this challenge, precautionary approaches to mitigating anthropogenic greenhouse gases will be necessary, while research continues on the full range of climate–health mechanisms and potential future health impacts.

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The role of increasing temperature variability in European summer heatwaves

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Instrumental observations[1,2] and reconstructions[3,4] of global and hemispheric temperature evolution reveal a pronounced warming during the past ~150 years. One expression of this warming is the observed increase in the occurrence of heatwaves[5,6]. Conceptually this increase is understood as a shift of the statistical distribution towards warmer temperatures, while changes in the width of the distribution are often considered small[7]. Here we show that this framework fails to explain the record-breaking central European summer temperatures in 2003, although it is consistent with observations from previous years. We find that an event like that of summer 2003 is statistically extremely unlikely, even when the observed warming is taken into account. We propose that a regime with an increased variability of temperatures (in addition to increases in mean temperature) may be able to account for summer 2003. To test this proposal, we simulate possible future European climate with a regional climate model in a scenario with increased atmospheric greenhouse-gas concentrations, and find that temperature variability increases by up to 100%, with maximum changes in central and eastern Europe.

A record-breaking heatwave affected the European continent in summer 2003. In a large area, mean summer (June, July and August, referred to as JJA below) temperatures have exceeded the 1961–90 mean by ~3 °C, corresponding to an excess of up to 5 standard deviations (Fig. 1a). Even away from the centre of action, many long-standing temperature records have tumbled.

For further analysis, we consider long-term temperature series from Switzerland, located close to the centre of the anomaly. Twelve carefully homogenized series[8,9] are available with daily resolution since 1864. To minimize contamination by local meteorological and instrumental conditions, we amalgamate four independent and particularly reliable stations (Basel-Binningen, Geneva, Bern-Liebefeld, and Zürich) into one single series with monthly temporal resolution. This series is representative for the northwestern foothills of the Alps. Figure 1b–e displays the statistical distribution of monthly and seasonal temperatures. The year 2003 is far off the distribution in three of the four panels. For instance, the previous record holder for JJA was 1947 with a temperature anomaly of $T' = 2.7$ °C (with respect to the 1864–2000 mean). The corresponding value for 2003 is as high as $T' = 5.1$ °C and this amounts to an offset of 5.4 standard deviations from the mean (the corresponding values of individual months are listed in Fig. 1). Such extreme values (which indeed have the characteristics of outliers) pose serious challenges to any analysis, as the statistical distribution so far away from the mean is not described by the data.

In a first step, we thus restrict attention to the time period 1864–2000 and compile compound statistics for all monthly temperature anomalies (January–December). The purpose of this is to identify changes near the tails of the statistical distribution that result from the warming trend in the series. To this end, we consider two 60-yr periods, one covering the beginning of the series (1864–1923), and one the end (1941–2000). Figure 2a, b shows the resulting statistical distributions, both in terms of cumulative probability and probability density functions. The two distributions show similar characteristics in general, but the 1941–2000 distribution is shifted by the mean warming ($\Delta T = 0.8$ °C) between the two periods. This shift also implies a change in the frequency of extremes. For instance (Fig. 2c), the frequency of a month with an anomaly of $T' = 3$ °C has increased by ~100%. Hence, a month in the 1941–2000 period with an excess temperature of $T' = 3$ °C can be tied with a probability of 50% to the warming between the two periods, in a probabilistic sense as recently proposed[10]. This illustrates how comparatively small shifts in climate mean may imply pronounced changes at the tails of the statistical distribution and in the frequency of extremes.

The dashed and full curves in Fig. 2 relate to the empirical and the fitted gaussian distributions, respectively, and their close agreement shows that the gaussian distribution is an excellent approximation to the data. The small reduction in variability (Fig. 2b) is not statistically significant, and is entirely due to changes in the month of December (where the variability was substantially reduced owing to the absence of cold northeasterly weather types).

A conclusive analysis such as that in Fig. 2 is not feasible for summer 2003, as there is only one data point so far off the mean. To quantitatively assess the situation, we have estimated its return

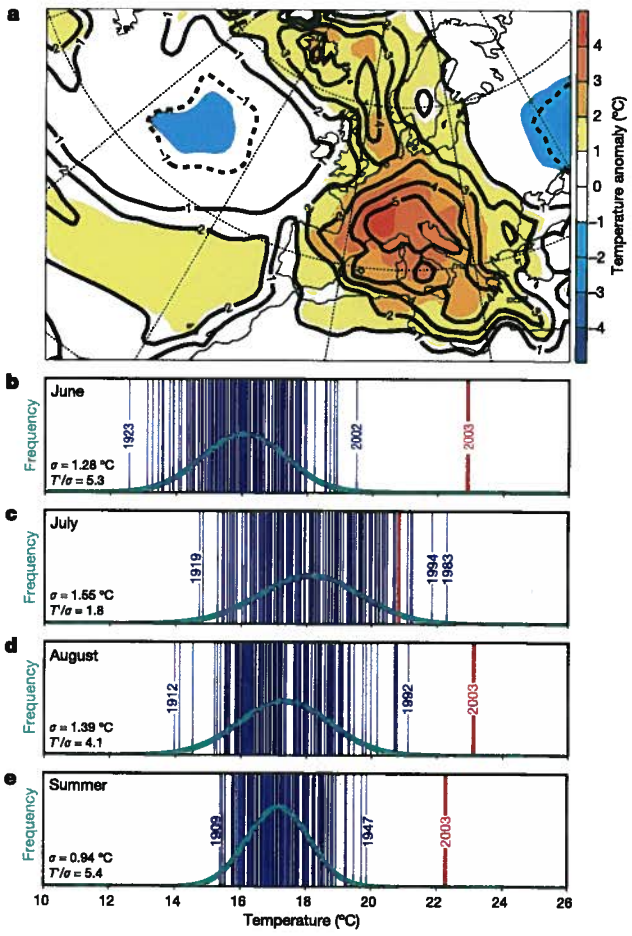

Figure 1 Characteristics of the summer 2003 heatwave. **a**, JJA temperature anomaly with respect to the 1961–90 mean. Colour shading shows temperature anomaly (°C), bold contours display anomalies normalized by the 30-yr standard deviation. **b–e**, Distribution of Swiss monthly and seasonal summer temperatures for 1864–2003. The fitted gaussian distribution is indicated in green. The values in the lower left corner of each panel list the standard deviation (σ) and the 2003 anomaly normalized by the 1864–2000 standard deviation (T'/σ). See Methods section for further details.

period. The return period τ is an estimate of the frequency of a particular event (or its exceedance) based on a stochastic concept. Here we employ a gaussian distribution fitted to JJA temperatures to estimate τ with respect to a selected reference period (see Methods section for details). With respect to the reference period 1864–2000, a return period of several million years is obtained, but such an excessive estimate based on a short series is dubious. To account for the warming in the last decades, we use a more recent reference period 1990–2002 (with $\Delta T = 1.25\,^{\circ}\mathrm{C}$ warmer mean temperature, but assuming an unchanged standard deviation). With respect to this climatology, the resulting return period for summer 2003 still amounts to $\tau = 46{,}000$ yr. The uncertainty of this estimate is considerable, however, and the lower bound of the 90% confidence interval is $\tau = 9{,}000$ yr.

This large return period should not be overstated, and is here merely used to express the rareness of such an extreme summer with respect to the long-term instrumental series available. In particular, the analysis does not exclude the possibility that such warm summers might have occurred in the more distant historical past, for instance in the Medieval Warm Period[11], in 1540[12,13] or in 1757. It suggests, however, that an event like summer 2003 does not fit into the gaussian statistics spanned by the observations of the reference period, but might rather be associated with a transient change of the statistical distribution. This interpretation is consistent with the idea that small changes of the statistical distribution can yield pronounced changes in the incidence of extremes[7,14].

As a shift of the statistical distribution by the observed mean warming is unable to explain the record-breaking summer 2003, we hypothesize that the heatwave might be due to a change of the distribution's width, representing an increase in year-to-year variability. Support for this hypothesis comes from a regional climate model (RCM) driven by a greenhouse-gas scenario representing 2071–2100 conditions (SCEN). The scenario integration is compared against a control integration covering the period 1961–90 (CTRL). At the lateral boundaries, the RCM is driven by a model chain consisting of two general circulation models (GCMs; see Methods section for details). The use of a high-resolution RCM increases our ability to compare the results against observations. The statistical temperature distribution for CTRL agrees notably well with observations. For the grid point in northern Switzerland, the summer climate is characterized by a temperature mean of $\bar{T} = 16.1\,^{\circ}\mathrm{C}$ and a standard deviation of $\sigma = 0.96\,^{\circ}\mathrm{C}$, while the long-term characteristics of our temperature series are $\bar{T} = 16.9\,^{\circ}\mathrm{C}$ and $\sigma = 0.94\,^{\circ}\mathrm{C}$.

Figure 3a, b displays JJA temperatures for the two integrations for a grid point in northern Switzerland. In the SCEN simulation, the distribution is shifted by ~4.6 °C towards warmer temperatures. More important, SCEN also exhibits a pronounced widening of its statistical distribution, with the standard deviation increasing by 102%. This widening is statistically highly significant ($P < 1\%$) and

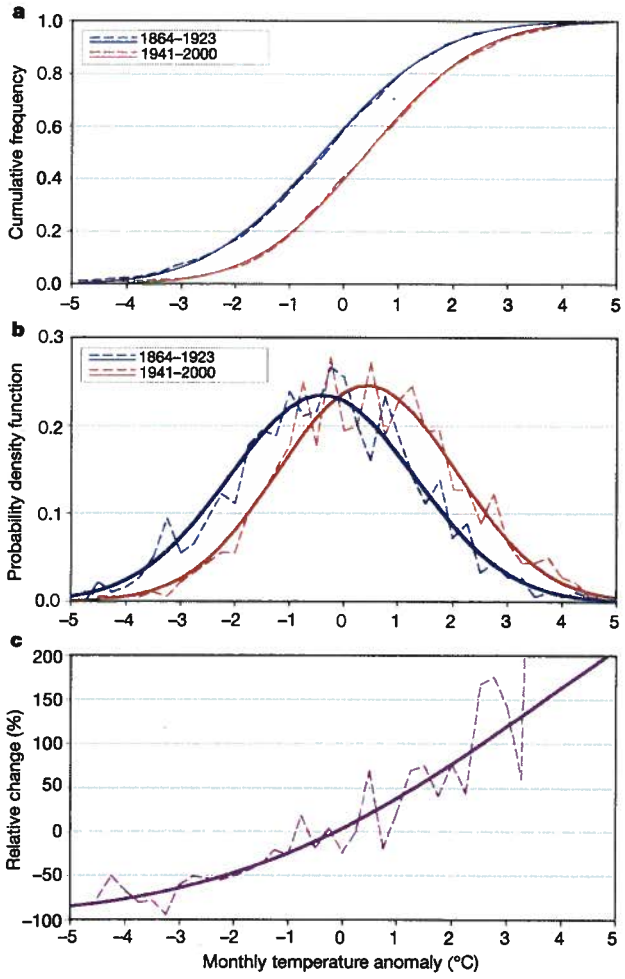

Figure 2 Statistical distribution of Swiss monthly temperature anomalies (compound statistics using January–December monthly data). Data in **a** and **b** are shown for the periods 1864–1923 (blue curves) and 1941–2000 (red curves). Panels show the cumulative frequency distribution (**a**), the probability density function (**b**), and the relative frequency change between the two periods (**c**). Full lines show the fit with the gaussian distribution, dashed lines are obtained from raw data.

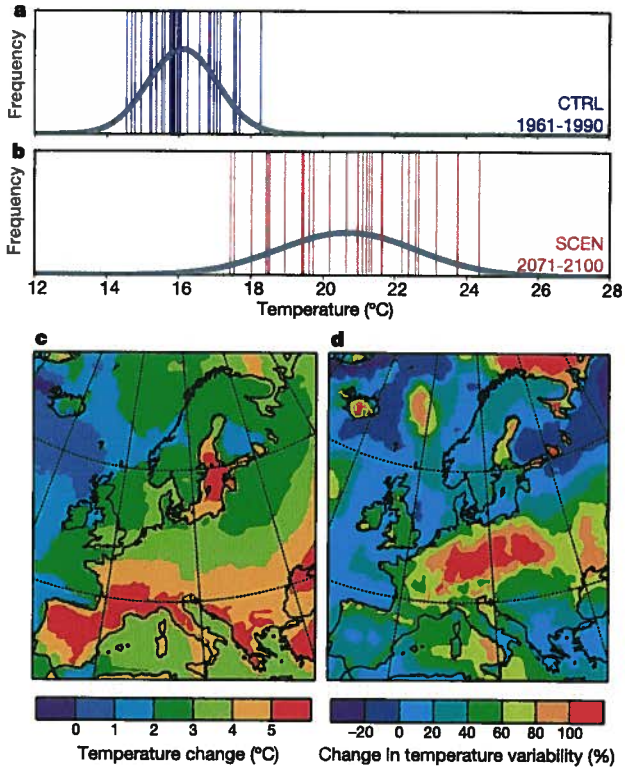

Figure 3 Results from an RCM climate change scenario representing current (CTRL 1961–90) and future (SCEN 2071–2100) conditions. **a**, **b**, Statistical distribution of summer temperatures at a grid point in northern Switzerland for CTRL and SCEN, respectively. **c**, Associated temperature change (SCEN–CTRL, °C). **d**, Change in variability expressed as relative change in standard deviation of JJA means ((SCEN–CTRL)/CTRL, %).

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only slightly affected by the transient warming within the two periods (a revised estimate using detrended temperature series yields a somewhat smaller variability increase of 86%). The spatial distribution of the relative increase in variability (Fig. 3d) shows a pronounced signal throughout central and eastern Europe that is not directly linked to the simulated mean temperature change (Fig. 3c). More detailed analysis suggests that the warm summers of SCEN show signs of drought[15], with the semi-arid Mediterranean climate progressing towards central Europe. In SCEN, central Europe is more often (but not always) affected by summer droughts than in CTRL, and this implies an increase in variability. The drought conditions develop in response to large-scale anticyclonic forcing, and they nonlinearly amplify local temperature anomalies. During droughts the net balance of solar and infrared radiation is almost entirely balanced by local heating, while evapotranspiration is suppressed owing to the lack of soil moisture[16]. This process may be further amplified by a positive feedback between soil moisture and precipitation[17,18].

The sequence of feedbacks involves substantial uncertainties due to large-scale anticyclonic forcing[19], radiation[20], soil hydrology[21] and other processes, which are difficult to represent in climate models. To check on our simulations, we have analysed other GCM and RCM scenarios of greenhouse-gas conditions, and find that all of these exhibit a substantially increased level of variability over large parts of Europe (we have studied one GCM and four RCM simulations from the PRUDENCE project; http://prudence.dmi.dk).

The simulated increase in variability also implies an increase in extremes relative to mean climatic conditions. For illustration, a 50% increase in the standard deviation of our long-term JJA temperature series ($\sigma = 0.94\,^{\circ}C$) would raise the probability of a 2003-like event ($T' = 3.85\,^{\circ}C$ with respect to 1990–2002) by a factor of ~150. For an event with $T' = 5\,^{\circ}C$, it would increase by a factor of ~5,100. This tremendous sensitivity of extremes to the width of the statistical distribution has led to the statement "variability is more important than averages"[14]. A recent increase in variability is thus a plausible hypothesis to explain extreme JJA 2003 conditions. Such a hypothesis would also be compatible with the occurrence of drastically different European summers such as in 2002 and 2003, but at present there are insufficient data to draw any firm conclusions.

To conclude, we address the question of whether the abnormal summer 2003 shows similar characteristics to those simulated in the RCM runs. To this end, summer temperature and precipitation anomalies are displayed against each other, both for the observations (Fig. 4a) and for the climate change simulations (Fig. 4b). Both panels include a data point representing observed JJA 2003 conditions, and the results apply to northern Switzerland. The observed data (Fig. 4a) are based on averages of conventional temperature and precipitation (rain-gauge) observations at the four stations referred to above, while the simulated data (Fig. 4b) are shown for a single grid point roughly corresponding to the location of our long-term series.

Several inferences can be drawn from the analysis. First, both data sets exhibit a similar (statistically significant) relationship between temperature and precipitation anomalies. The regression analysis yields slopes of $-11\%\,^{\circ}C^{-1}$ and $-8.2\%\,^{\circ}C^{-1}$ for the observations and the simulations, respectively. Thus, although there is some underestimation of summer precipitation in CTRL (at the grid point under consideration, by 21%), the simulations credibly represent the observed precipitation sensitivity. Despite a general trend towards drier conditions with increasing temperatures, there is also an increase in the incidence of heavy precipitation events[22].

Second, Fig. 4b demonstrates that in terms of temperature and precipitation the climatic conditions in JJA 2003 were not unlike those simulated by SCEN for the period 2071–2100. For northern Switzerland, the 2003 observation is located approximately in the middle of the SCEN data points (Fig. 4b). Thus, the RCM simulations suggest that towards the end of the century—under the given scenario assumptions—about every second summer could be as warm or warmer (and as dry or dryer) than 2003.

Our results demonstrate that the European summer climate might experience a pronounced increase in year-to-year variability in response to greenhouse-gas forcing. Such an increase in variability might be able to explain the unusual European summer 2003, and would strongly affect the incidence of heatwaves and droughts in the future. It would represent a serious challenge to adaptive response strategies designed to cope with climate change. □

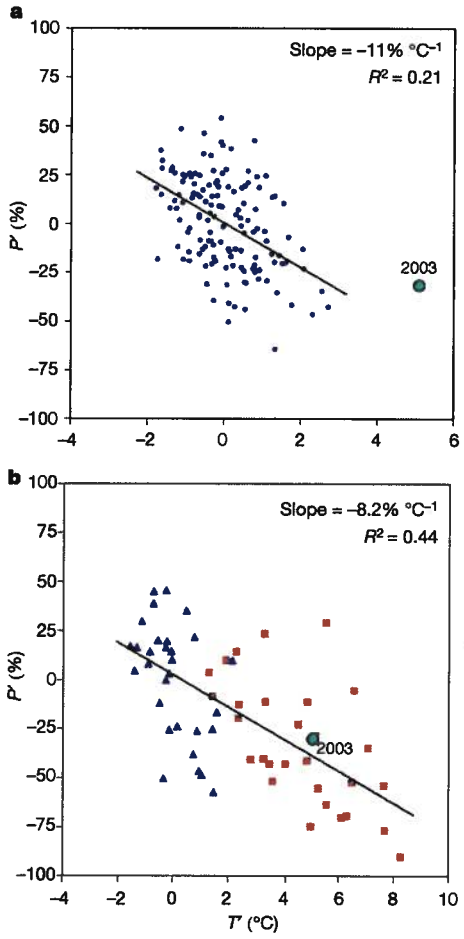

Figure 4 Scatter diagrams showing summer mean temperature and precipitation anomalies for northern Switzerland. **a**, Long-term (1864–2003) station data with respect to 1961–90 means. **b**, Climate change simulations CTRL (1961–90, blue symbols) and SCEN (2071–90, red symbols) with respect to CTRL means. The green symbols show the observations for JJA 2003. The regression lines in **a** and **b** are based on 1864–2002 data and combined CTRL and SCEN data, respectively.

Methods

Large-scale analysis of summer 2003

The continental-scale temperature anomaly for JJA 2003 (Fig. 1a) is based on ERA-40 reanalysis data[23] (for 1961–90) and operational meteorological analysis data (for 2003) of the European Centre for Medium-Range Weather Forecasts (ECMWF; see http://www.ecmwf.int). Monthly temperatures are computed as means of daily T_{min} and T_{max}. Small height differences between the ERA-40 and ECMWF topographies are accounted for by the use of an adiabatic lapse rate (0.6 °C per 100 m).

Estimation of return period

The stochastic concept adopted in the estimation of return periods assumes independent, identically distributed JJA temperatures with the underlying distribution being gaussian.

The distribution parameters are estimated from the data of the reference period, using the method of moments (which in the case of a gaussian distribution is identical to maximum-likelihood estimation). The return period of the event (expected frequency of threshold exceedance) is then calculated from the fitted distribution. Confidence bounds of the return period were calculated by parametric resampling. These take into account the uncertainty of the parameter estimates given the finite sample size (that is, the number of summers in the reference period), but not the uncertainty in the underlying stochastic concept. We have also tested whether the data are reasonably gaussian distributed, checking quantile-quantile plots (see also Fig. 2a).

Climate change simulations

The climate change scenario is based on the SRES A2 transient greenhouse-gas scenario as specified by the Intergovernmental Panel on Climate Change (IPCC)[24]. The scenario computations involve three numerical models: the low-resolution HadCM3 global coupled atmosphere–ocean GCM, the intermediate-resolution HadAM3H atmospheric GCM, and the CHRM limited-area high-resolution RCM. The HadCM3 simulation[25,26] is a long integration using the observed atmospheric composition for 1859–1990 and scenario conditions for 1991–2100. For the HadAM3H simulation[27], two time-slice experiments are available, representing control (1961–90) and scenario (2071–2100) conditions. The former is driven by observed sea surface temperature and sea-ice distributions, while the latter uses the changes from the HadCM3[28]. The CHRM RCM[29] is used with a horizontal resolution of 56 km and 20 levels in the vertical, and is driven at its lateral boundaries by HadAM3H. The CHRM has been validated regarding its ability to represent observed natural interannual variations[29]. The simulated increase in temperature variability in SCEN is largely determined by the soil hydrology of the model under consideration. For instance, it is substantially smaller in the CHRM than in the driving HadAM3H simulation.

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Intensification of hot extremes in the United States

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Abstract

Governments are currently considering policies that will limit greenhouse gas concentrations, including negotiation of an international treaty to replace the expiring Kyoto Protocol. Existing mitigation targets have arisen primarily from political negotiations, and the ability of such policies to avoid dangerous impacts is still uncertain. Using a large suite of climate model experiments, we find that substantial intensification of hot extremes could occur within the next 3 decades, below the 2 °C global warming target currently being considered by policy makers. We also find that the intensification of hot extremes is associated with a shift towards more anticyclonic atmospheric circulation during the warm season, along with warm-season drying over much of the U.S. The possibility that intensification of hot extremes could result from relatively small increases in greenhouse gas concentrations suggests that constraining global warming to 2 °C may not be sufficient to avoid dangerous climate change.

1. Introduction

World governments are currently considering mitigation policies that will limit greenhouse gas (GHG) concentrations, including an international treaty to replace the expiring Kyoto Protocol [*UNFCCC*, 2009]. Key questions include the level of GHG forcing that should be targeted and the urgency with which that target should be achieved, with considerable discussion oriented around trade-offs between avoiding policy-induced economic damage and GHG-induced climate damage (e.g., [*Mastrandrea and Schneider*, 2004]). However, existing mitigation targets – such as the target of 2 °C global warming above pre-industrial conditions set by world governments as part of the recent Copenhagen Accord [*UNFCCC*, 2009] – have arisen primarily from political negotiations. Although substantial scientific work has focused on the climate system response to varying GHG concentrations [*Mastrandrea and Schneider*, 2004;

Meehl et al., 2007b], there remains uncertainty as to whether "dangerous" climate change impacts could emerge below the target GHG envelope currently being considered by policy makers.

The hot extremes that are an important source of potential climate change impacts (e.g., [*Battisti and Naylor*, 2009; *Poumadere et al.*, 2005]) can result from both large- and fine-scale climate processes. For instance, the 2003 European heat wave was associated with large-scale anticyclonic atmospheric anomalies [*Meehl and Tebaldi*, 2004], with local and regional land coupling both enhancing the large-scale circulation anomalies and accounting for more than half of the hot-day occurrence over much of the region [*Fischer et al.*, 2007b]. Likewise, the 20th-century Sahel drought has been attributed to a combination of large-scale ocean-atmosphere teleconnections and fine-scale land-atmosphere feedbacks [*Christensen et al.*, 2007]. Further, the response of hot extremes to high levels of GHG forcing appears sensitive to both large-scale atmospheric circulation and fine-scale surface-atmosphere interactions [*Diffenbaugh et al.*, 2005; *Meehl and Tebaldi*, 2004; *Seneviratne et al.*, 2006]. Quantification of the potential for near-term intensification of hot extremes therefore requires a climate modeling framework that can capture the uncertainties associated with both large- and fine-scale climate processes.

2. Methods

We employ the RegCM3 nested climate model [*Pal et al.*, 2007], using the grid of [*Diffenbaugh et al.*, 2005], which covers the continental U.S. at 25-km horizontal resolution and 18 levels in the vertical. Our transient experiment includes five members simulating the period from 1950 to 2039 in the A1B emissions scenario [*IPCC*, 2000]. The first year (1950) is discarded to account for model equilibration. Each RegCM3 ensemble member uses the same

parameterization options (as in [*Diffenbaugh et al.*, 2005]), with only the large-scale input varying between the members.

Large-scale boundary conditions are provided by the NCAR CCSM3 [*Collins et al.*, 2006]. We use five of the CCSM3 simulations archived as part of the CMIP3 intercomparison [*Meehl et al.*, 2007a]. (These CCSM3 ensemble members are identified by NCAR as c, e, bES, fES, and gES.) In order to generate the necessary sub-daily, 3-dimensional atmospheric variables, we re-run the atmospheric component (CAM3) from 1948 to 2039, using the original CCSM3-generated SSTs and sea ice as boundary conditions for the global atmosphere (see [*Trapp et al.*, 2009]). These CAM3 simulations use the same resolution as in the original CCSM3 simulations (T85 spectral truncation with 26 levels in the vertical). We also analyze GCM output from the CMIP3 climate model archive [*Meehl et al.*, 2007a], selecting the output from "run 1" of each of the 22 GCMs that archived monthly surface air temperature results for the A1B scenario.

We first calculate the hottest season of the 1951-1999 period at each grid point. Both the CMIP3 and RegCM3 ensembles are able to capture the observed magnitude and pattern of hottest-season and mean-summer temperature in the U.S. (Fig. S1). The simulation of interannual variance of summer temperature is less accurate, with over-estimation of variance in the central U.S. (Fig. S1). ([*Walker and Diffenbaugh*, 2009] diagnose the RegCM3 warm-season temperature biases over the U.S., including biases in the atmospheric circulation and moisture.) For each 21st century model realization, we calculate the number of exceedences of the hottest season of the respective 1951-1999 period. We then calculate the ensemble mean and standard deviation across the respective ensemble members.

In addition, because of the availability of sub-daily output from the RegCM3 realizations, we are also able to calculate the occurrence of the annual-scale 95th-percentile daily maximum temperature (T95), and of the longest historical heat wave. For the former, which quantifies the frequency of exceedence of the present tail of the daily temperature distribution, we follow [*Diffenbaugh et al.*, 2005], using the 1980-1999 period as a baseline. In this approach, the T95 threshold at each grid point is calculated as the mean of the daily maximum temperature values from the 18th hottest day of each year in the baseline period. For the latter, we apply the heat wave duration index of [*Frich et al.*, 2002] to find the longest heat wave of the 1951-1999 period, along with the 21st century occurrence of heat waves that are at least as long as this historical maximum. As with the historical hottest season exceedence, we calculate the baseline and exceedence values at each grid point, and for each decade of the 2010-2039 period.

3. Results

We find that the exceedence of the historical hottest-season threshold increases over the next three decades in the A1B scenario (Fig. 1). The intensification of hot extremes emerges quickly in the RegCM3 simulations, with 3 to 4 exceedences per decade over large areas of the U.S. in the 2010-2019 period (Fig. 1) (with an intra-ensemble standard deviation (S.D.) of 2 to 3 exceedences per decade over most of the U.S.; Fig. S2). This emergence intensifies in the 2020-2029 period, with up to 8 exceedences per decade over the western U.S. (S.D. of 3 to 4), and up to 4 exceedences per decade over much of the eastern U.S (S.D. of 2 to 3). Further, in the 2030-2039 period, most areas of Utah, Colorado, Arizona and New Mexico experience at least 7 exceedences per decade (S.D. of 3 to 4), and much of the rest of the U.S. experiences at least 4 exceedences per decade (S.D. of 2 to 5 over most areas). The summer warming in the RegCM3 ensemble is not uniform, with greater increases in the mean in the eastern U.S. than the western

U.S. (Fig. S3), along with increased variance in the northcentral U.S., increased skewness in the southwestern and southeastern U.S., and decreased kurtosis throughout most of the continental U.S. (Fig. S3). The intensification of hottest-season exceedence is similar in the CMIP3 ensemble (compared with the RegCM3 ensemble), including up to 6 exceedences per decade over the western and northeastern U.S. in the 2030-2039 period, and up to 8 exceedences per decade over parts of the southeastern U.S. (with S.D. of 4 over most of the western and eastern U.S., and 3 over most of the central U.S.). However, the intensification of seasonal hot extremes emerges more quickly and strongly in the RegCM3 ensemble, particularly over the western U.S., where the higher-resolution topographic boundary condition leads to a more accurate representation of extreme seasonal temperature values (Fig. S1).

The annual occurrence of the T95 threshold exceeds 30 days per year over much of the U.S. during the 2020-2029 period (Fig. 1) (S.D. of 2 to 12; Fig. S2), with peak occurrence of up to 52 days per year over Texas and Florida (S.D. of 10 to 24). T95 occurrence exceeds 38 days per year over much of the U.S. in the 2030-2039 period (S.D. of 4 to 16), with the area exceeding 46 days per year expanding to include most of the southern Great Plains and much of the Gulf Coast region (S.D. of 10 to 24). Likewise, the area experiencing at least one exceedence of the historical heat wave threshold per decade covers most of the U.S. in the 2020-2029 period, including up to 5 exceedences per decade over areas of the western and central U.S. (Fig. 1) (S.D. of 1 to 5 over most of the U.S.; Fig. S2). Occurrence of the longest historical heat wave further intensifies in the 2030-2039 period, including greater than 5 occurrences per decade over much of the western U.S., and greater than 3 exceedences per decade over much of the eastern U.S. (S.D. of 3 to 7 over most of the U.S.).

4. Discussion

The intensification of hot extremes in the RegCM3 ensemble is associated with warm-season drying over much of the U.S. (Fig. 2). By the 2030-2039 period, a summer anticyclonic circulation anomaly develops aloft (at 500 mb) over most of the continental U.S. Associated with this anticyclonic anomaly are decreases (2030-2039 minus 1980-1999) in precipitation (exceeding -1.0 mm/day), total soil moisture (exceeding -125 mm), and evapotranspiration (exceeding -0.6 mm/day). Although the large-scale circulation anomalies are very similar between the driving CAM3 and nested RegCM3 fields in the autumn, winter and spring, the summer anticyclonic anomaly is more widespread in RegCM3 than CAM3 (Fig. S4).

We find that the coupling of changes in summer temperature, precipitation and soil moisture is robust across the model realizations. For the 2030-2039 period, all five RegCM3 members exhibit a negative correlation between changes in summer total soil moisture and changes in summer temperature, and a positive correlation between changes in summer total soil moisture and changes in summer precipitation (Fig. 3). (The ensemble mean correlation is -0.35 for change in temperature and 0.37 for change in precipitation.) We also find that all five RegCM3 members exhibit a decrease in summer total soil moisture across the domain. (The ensemble mean fractional change in total soil moisture is -0.02.) For the CMIP3 ensemble, we find that 89% of the realizations show a negative (positive) correlation between changes in summer soil moisture and changes in summer temperature (precipitation). (The ensemble mean correlation is -0.28 for change in temperature and 0.30 for change in precipitation.) We also find that 78% of the GCM realizations show a decrease in summer total soil moisture across the domain for the 2030-2039 period. (The ensemble mean fractional change in total soil moisture is -0.03.)

Surface drying associated with anticyclonic circulation anomalies is thought to have amplified severe hot and dry events such as the 1988 event in the U.S. [*Chen and Newman*, 1998] and the 2003 event in Europe [*Fischer et al.*, 2007b], and has been identified as a key regulator of changes in climate variability in response to elevated GHG forcing [*Seneviratne et al.*, 2006]. The fact that most of the GCM realizations simulate soil-moisture/temperature/precipitation relationships of the same sign as the RegCM3 ensemble suggests that the coupling is likely to be robust over the U.S., a result that supports previous work (e.g., [*Fischer et al.*, 2007a; *Lorenz et al.*, 2010; *Seneviratne et al.*, 2006]). However, although we have identified correlations between changes in temperature, precipitation, and soil moisture that are robust across a large suite of climate model experiments, it is not clear from the analysis of these experiments alone whether the surface drying is the cause of the intensified hot extremes. For instance, the decreases in soil moisture could be a product of decreases in precipitation (Fig. 2) and/or increases in net surface radiation (Fig. S5) associated with the changes in large-scale circulation (Fig. 2). Targeted experiments that physically isolate moisture fluxes, radiation fluxes, and atmospheric circulation (as in [*Seneviratne et al.*, 2006]) are necessary in order to fully determine causation.

The spread within the CMIP3 ensemble (in which multiple GCMs are included) is greater than the spread within the RegCM3 ensemble (in which only one GCM-RCM combination is included) (Fig. S2, 3). Earlier work using an RCM nested within an atmosphere-only GCM suggests that some of the spread in our nested ensemble could be generated by internal atmospheric variability [*Dutton and Barron*, 2000]. The fact that our high-resolution ensemble is nested within an ensemble of coupled AOGCM experiments further enhances the effects of internal variability on the ensemble simulation. This atmosphere-ocean internal variability

dominates the near-term "uncertainty" in the CMIP3 ensemble [*Hawkins and Sutton*, 2009]. However, by the mid-century, structural uncertainty from different model formulations is greater than that from internal variability [*Hawkins and Sutton*, 2009], suggesting that multiple GCM-RCM combinations could yield greater spread than is seen in our nested simulations.

Our results suggest that near-term increases in GHG forcing could result in warm-season drying and intensification of hot extremes throughout much of the U.S. Indeed, all of the individual RegCM3 ensemble members exhibit at least 6 hottest-season occurrences in the 2030-2039 period over much of the western U.S. (Fig. S6). However, the members vary in the level of hot event intensification in the eastern U.S., with three of the members showing substantial intensification in the 2030-2039 period, and two of the members showing very little intensification (Fig. S6). (For reference, the RegCM3 f-member shows the greatest summer warming over the continental U.S. in the 2030-2039 period, while the g-member shows the least.) The variation seen within the physically-uniform RegCM3 ensemble (Fig. S2, S3 and 3) suggests a strong influence of internal variability on decadal-scale changes in regional- and local-scale hot extremes.

5. Conclusions

Because of the known sensitivity of natural and human systems, intensification of hot extremes could carry substantial impacts. At the end of the 2030-2039 period, the expected global mean temperature change relative to the late 20th century ranges from 1.0 to 1.7 °C in the CMIP3 A1B scenario [*Meehl et al.*, 2007b], and from 1.1 to 1.3 °C in the CCSM3 ensemble [*Meehl et al.*, 2006]. Given the IPCC calculation of approximately 0.8 °C of global warming from the mid-19th century to the late 20th century [*Trenberth et al.*, 2007], the CMIP3 ensemble warming above pre-industrial conditions is approximately 1.8 to 2.5 °C by the year 2040, while

the CCSM3 ensemble is approximately 1.9 to 2.1 °C. Further, given that global warming is likely to continue for decades after stabilization of GHG concentrations [*Meehl*, 2005], and that the late-21st century warming in the A1B scenario ranges from 2.25 to 4.25 °C above the late 20th century [*Meehl et al.*, 2007b], the response to a given GHG stabilization target is likely to be greater than to the equivalent concentrations within the transient trajectory tested here. Although accurate decadal-scale climate prediction represents a significant challenge (e.g., [*Meehl et al.*, 2009]), the intensification of hot extremes reported here suggests that constraining global warming to 2 °C above pre-industrial conditions may not be sufficient to avoid dangerous climate change.

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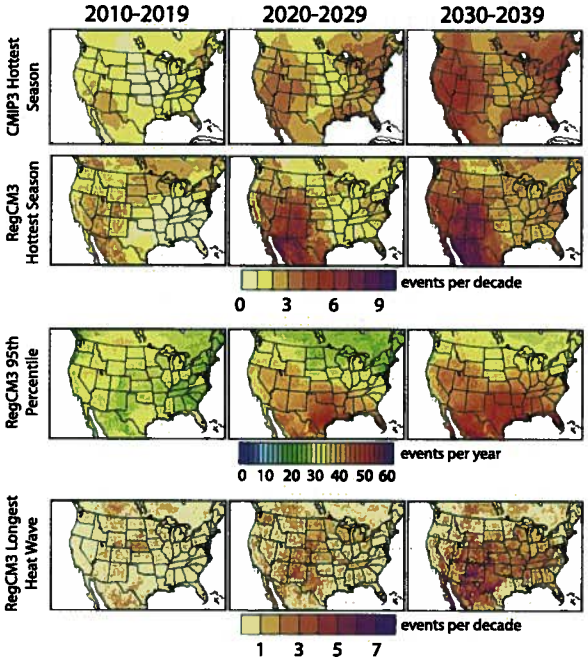
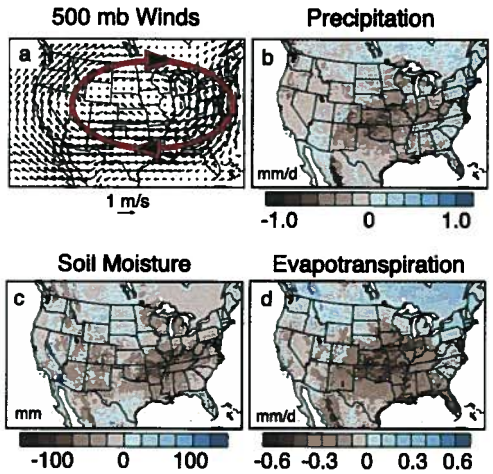
8. Figure Captions

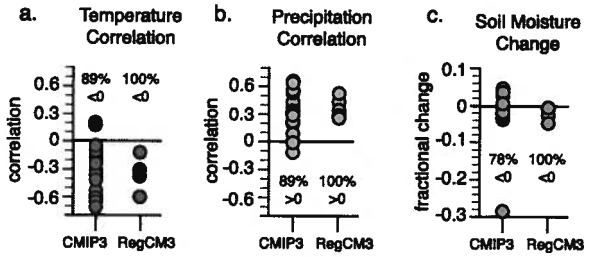
Figure 1. Projected changes in heat extremes in the coming decades. The top two rows show the decadal occurrence of the 1951-1999 hottest-season threshold in the CMIP3 and RegCM3 ensembles. The third and fourth rows show the decadal occurrence of the 95^{th}-percentile daily maximum threshold (T95) and the historical hottest-heat-wave threshold for the RegCM3 ensemble.

Figure 2. Changes in summer 500 mb winds (a), precipitation (b), total soil moisture (c), and evapotranspiration (d) in the RegCM3 ensemble. Changes are calculated as 2030-2039 minus 1980-1999 for June-July-August. The ellipse and large arrows in panel (a) are added for emphasis.

Figure 3. Simulated relationships between summer temperature, precipitation, and soil moisture in the RegCM3 and CMIP3 ensembles. (a) Correlation between the change in summer temperature and the change in summer soil moisture for the 2030-2039 period. (b) Correlation between the change in summer precipitation and the change in summer soil moisture for the 2030-2039 period. (c) Change in summer soil moisture for the 2030-2039 period. Each circle represents one model realization. We first calculate the change in mean summer temperature, precipitation and soil moisture for the 2030-2039 period (relative to the 1980-1999 period, with the change in total soil moisture calculated as a fraction of the 1980-1999 summer mean). We then calculate the correlation between the change in temperature (precipitation) and the change in soil moisture across the land grid points that encompass the RegCM3 domain. The CMIP3 ensemble uses "run 1" from the 18 CMIP3 GCMs archiving total soil moisture.


2010-2019
2020-2029
2030-2039
CMIP3 Hottest Season
RegCM3 Hottest Season
events per decade
0 3 6 9
RegCM3 95th Percentile
events per year
0 10 20 30 40 50 60
RegCM3 Longest Heat Wave
events per decade
1 3 5 7


500 mb Winds
Precipitation
a
b
1 m/s
mm/d
-1.0
0
1.0
Soil Moisture
Evapotranspiration
c
d
mm
mm/d
-100
0
100
-0.6
-0.3
0
0.3
0.6


a.
Temperature Correlation
correlation
0.6
0.3
0
-0.3
-0.6
89% ≤0
100% ≤0
CMIP3
RegCM3
b.
Precipitation Correlation
correlation
0.6
0.3
0
-0.3
-0.6
89% >0
100% >0
CMIP3
RegCM3
c.
Soil Moisture Change
fractional change
0.1
0
-0.1
-0.2
-0.3
78% ≤0
100% ≤0
CMIP3
RegCM3

Climatic Change
DOI 10.1007/s10584-006-9166-7

Climate change, ambient ozone, and health in 50 US cities

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Abstract We investigated how climate change could affect ambient ozone concentrations and the subsequent human health impacts. Hourly concentrations were estimated for 50 eastern US cities for five representative summers each in the 1990s and 2050s, reflecting current and projected future climates, respectively. Estimates of future concentrations were based on the IPCC A2 scenario using global climate, regional climate, and regional air quality models. This work does not explore the effects of future changes in anthropogenic emissions, but isolates the impact of altered climate on ozone and health. The cities' ozone levels are estimated to increase under predicted future climatic conditions, with the largest increases in cities with present-day high pollution. On average across the 50 cities, the summertime daily 1-h maximum increased 4.8 ppb, with the largest increase at 9.6 ppb. The average number of days/summer exceeding the 8-h regulatory standard increased 68%. Elevated ozone levels correspond to approximately a 0.11% to 0.27% increase in daily total mortality. While actual future ozone concentrations depend on climate and other influences such as changes in emissions of anthropogenic precursors, the results presented here

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indicate that with other factors constant, climate change could detrimentally affect air quality and thereby harm human health.

1 Introduction

Climate change could harm human health in many ways, including adverse changes in food production (Rosenzweig et al. 2001), malaria (Loevinsohn 1994; Tanser et al. 2003), dengue fever (Hales et al. 2002), thermal stress (Martens 1998), aeroallergens (Beggs 2004), extreme events (Ikeda et al. 2005; Knutson et al. 1998), waterborne diseases (Casman et al. 2001; Charron et al. 2004), and other diseases (Epstein 2001; Hunter 2003; Patz et al. 2005; Reiter 1998). Changes in climate could also affect health by increasing the concentrations of outdoor air pollutants (Bernard et al. 2001; Haines and Patz 2004; Knowlton et al. 2004; McMichael and Githeko 2001). A recent study of ozone-related health impacts from climate change in 31 counties in New York projected a median 4.5% increase in ozone-related acute summer mortality by the 2050s, as compared to the 1990s (Knowlton et al. 2004). Tropospheric ozone is particularly sensitive to climate change because the chemical reactions that form ozone are temperature dependent, with higher levels of ozone produced during warmer time periods (Aw and Kleeman 2003; Seinfeld and Pandis 2006; Sillman and Samson 1995). Interannual variability in ozone levels is related to summer weather conditions for this region. For example, low ozone levels during the summer of 2003 were largely attributable to favorable (cool and wet) weather conditions across much of the United States (USEPA 2004). Further, biogenic emissions of volatile organic compounds (VOCs), which are precursors to ozone, increase with rising temperature (Constable et al. 1999). These natural emissions of VOCs constitute a significant fraction of total VOCs, and in some areas exceed anthropogenic sources (Fuentes et al. 2000).

Ozone levels have generally declined in the United States since the enactment of the Clean Air Act in 1970 due to emission control programs. However, high ozone concentrations persist with many areas still having levels above the health-based National Ambient Air Quality Standards (NAAQS). Currently, over 100 million people in the US reside in areas with ozone concentrations exceeding the 8-h regulatory standard (USEPA 2004). An increase in ozone concentrations, induced by climate change, would add to this already present health burden, which has been associated with higher levels of hospital admissions, respiratory symptoms, impaired lung development, and mortality, among other adverse health responses (Anderson et al. 2004; Bell et al. 2005; Dockery and Pope 1994; Gaudermann et al. 2002; Levy et al. 2001; Lippman 1989; Steib et al. 2002, 2003; Thurston and Ito 1999, 2001; USEPA 2006). Further, because of fossil fuel combustion, climate change control policies can have short-term benefits to local air quality, and thereby human health, in addition to their effects on long-term climate change and health (Bell et al. 2006; Cifuentes et al. 2001a,b).

We investigated how climate change could affect ozone levels and the subsequent changes in health impacts for 50 cities in the eastern US. A model of future climate change and a linked air pollution modeling system were used to compare ozone levels for current and potential future climatic conditions during the summer months (June, July, and August) for the 1990s and 2050s. In addition to meteorological variables such as temperature, wind speed, and wind direction that may change in a future climate, air quality conditions also depend on anthropogenic and biogenic emissions of ozone precursors, which in turn are a function of numerous factors such as population growth, energy demand, transportation

networks, land-use, fuel type, and pollution control technology. In order to explore the effect of climate change alone, this work isolates the response of tropospheric ozone concentrations to changes in climate, without regard to changes in anthropogenic emissions.

The human health consequences of ozone can be estimated through the change in health endpoints as identified by concentration-response functions from epidemiological studies. Other measures include exceedances of regulatory standards, such as the primary NAAQS, which were established to protect human health with an adequate margin of safety (USEPA 1997), and changes in the Air Quality Index (AQI), which is intended to give an overall assessment of the health impacts of a particular day's pollution levels (USEPA 2003). In this study, we utilize these measures to estimate the range of the potential human health consequences corresponding to altered ozone concentrations under a future climate scenario.

2 Methods

2.1 Estimates of ozone concentrations

Hourly ambient concentrations of ground-level ozone were estimated for each of 50 cities for June 1 to August 31 for 1993 to 1997 to represent the current climate, and for the summers of 2053 to 2057 to represent the future climate under the Intergovernmental Panel on Climate Change (IPCC) A2 scenario. The simulations account for climate change-induced alterations in the chemical reaction rates for tropospheric ozone formation and temperature-related changes in the emissions of biogenic ozone precursors (Hogrefe et al. 2004a), however the anthropogenic emission inventory is held constant. The climate and air quality simulations and the IPCC A2 emissions scenario are described in more detail below.

A linked climate/air quality modeling system developed by the New York Climate and Health Project (Hogrefe et al. 2004a,b; Knowlton et al. 2004) was used to derive ozone concentrations for each of 4,012 gridcells in each vertical layer in a domain with 36-km horizontal resolution for the eastern US. In this study, we utilize these simulations by interpolating the gridded concentration fields for the surface layer to the location of 50 cities in the eastern US. The modeling system included the Goddard Institute for Space Studies (GISS) general circulation model (GCM) (Russell et al. 1995), the PSU/NCAR mesoscale model (MM5) regional meteorological model (Grell et al. 1994), the Community Multiscale Air Quality (CMAQ) air quality model (Byun and Ching 1999), and the Sparse Matrix Operator Kernel Emissions (SMOKE) emissions processor (Houyoux et al. 2000). The climate effects of the A2 greenhouse gas (GHG) emissions scenario were simulated with the MM5 regional meteorological model driven by the GISS GCM; ozone pollution effects were simulated using the CMAQ air quality model for five summers in the 1990s; and model results were validated for current climate and air quality conditions (Grell et al. 1994; Hogrefe et al. 2004b; Russell et al. 1995).

Model evaluation included comparison of model estimates to observations for overall patterns and values and for variability. For example, evaluation of the meteorological model compared model-predicted meteorological fields, such as surface temperature, clouds, and winds, to observations. Comparisons used several approaches, including spectral decomposition, synoptic typing analysis, and use of cumulative distribution functions to estimate variability. The air quality model evaluation demonstrated that the model provides realistic estimates of the pattern of the daily maximum 1-h concentration over the 1993 to 1997 summers. The bias of estimated and observed concentrations was <1 ppb. The coupled MM5/

CMAQ modeling system produced reasonable estimates of mean ozone levels, signifying that emissions and synoptic-scale meteorology are well represented in the system (Hogrefe et al. 2004b). Further details on the modeling system, evaluation, and its application are available elsewhere (Hogrefe et al. 2004a,b; Lynn et al. 2004).

Climate and ozone concentrations for June, July, and August in the 2050s were simulated with the linked models. Projected future ozone concentrations were compared to those of the 1990s, holding constant all other human contributions to ozone pollution. Average summertime temperatures for the eastern US were projected to rise by 1.6 to 3.2 °C for these 50 cities from the 1990s to the 2050s.

The IPCC established several scenarios of GHG emissions for use in modeling studies. These emissions scenarios differ based on estimates of population, technology, economic growth, and other factors (Nakicenovic and Swart 2000). The modeling simulations analyzed in this study used the IPCC A2 climate scenario, one of the scenarios with the highest growth of carbon dioxide among all IPCC scenarios. This scenario is characterized by a steady increase in carbon dioxide emissions, a fuel mix determined by regional resource availability, a shift towards post-fossil fuel technologies in high-income but resource poor regions, reliance on older fossil fuel technology in lower-income resource-rich regions, and a worldwide population at 15 billion by 2100 (Nakicenovic and Swart 2000). This scenario assumes that decision makers place little emphasis on environmental concerns, however it does include control of GHGs that impact local air quality and controls on pollutants that affect water availability, soil quality, and agricultural productivity.

A complete picture of future ozone concentrations would require exploration of an extensive list of potential changes including regulatory controls, population growth, energy use patterns, and energy technologies. This work isolates the impact of climatic changes on ozone, and does not explore future changes in the emissions of anthropogenic precursors. In other words, this research investigates what modern day ozone levels might look like under a projected future climatic scenario. To fully gauge the range of potential ozone concentrations and related health consequences, a wide variety of scenarios would need to be explored.

2.2 Health and regulatory impacts

We calculated exceedances of the 1- and 8-h health-based regulatory standards for ozone, which differs from non-attainment of these standards (USEPA 1997). An exceedance day occurs when ozone levels go above the regulatory standard, and concentrations may be rounded down so that exceedances of the 1- and 8-h standards are above 124 and 84 parts per billion (ppb), respectively. 'Attainment' of the legal requirement is calculated using several years of data. In other words, an area could exceed the regulatory standard on a given day, but still be in attainment with the standard, depending on the concentrations of other days. However, an increase in the number of exceedance days would likely bring more areas into non-attainment status.

We also calculated what fraction of the summer days would fall under each category of the AQI for ozone. The AQI is used to provide an overall assessment of the health impacts of outdoor air with respect to several pollutants (USEPA 2003). The daily AQI is determined by assigning an individual index to each of several pollutants: ozone (8- and 1-h averages); particulate matter (PM_{10} and $PM_{2.5}$); carbon monoxide, sulfur dioxide, and nitrogen dioxide. The highest of these individual indices is assigned as the overall AQI for that day. Thus, while the AQI is representative of air pollutant levels, it does not provide an overall picture of air quality. The AQI can range from 0 to 500, with 0 representing the best air quality and 500 the worst. The AQI health-related levels for ozone are listed in Table 1.

Table 1 Air Quality Index (AQI) levels for ozone (modified from (USEPA 2003))

AQI	Ozone 8-h (ppb)	Ozone 1-h (ppb)	Air quality	Color code	Health advisory
0 to 50	0 to 64	0 to 84	Good	Green	None
51 to 100	65 to 84	85 to 124	Moderate	Yellow	Unusually sensitive people should consider limiting prolonged outdoor exertion
101 to 150	85 to 104	125 to 164	Unhealthy for sensitive groups	Orange	Active children and adults, and people with respiratory disease, such as asthma, should limit prolonged outdoor exertion
151 to 200	105 to 124	165 to 204	Unhealthy	Red	Active children and adults, and people with respiratory diseases, such as asthma, should avoid prolonged outdoor exertion; everyone else, especially children, should limit prolonged outdoor exertion
201 to 300	125 to 374	205 to 404	Very unhealthy	Purple	Active children and adults, and people with respiratory disease, such as asthma, should avoid all outdoor exertion; everyone else, especially children should limit outdoor exertion
301 to 500	375+	405+	Hazardous	Maroon	Everyone should avoid physical activity outdoors due to emergency pollution conditions

Health advisories in the form of ozone alert days are evaluated from forecasted ozone concentrations. An ozone 'advisory' is issued when the ozone forecast has an orange AQI, whereas an ozone 'alert' is declared when the ozone forecast has a red AQI. An ozone health 'alert' is issued for code purple, however this is a rare occurrence.

Concentration-response functions from epidemiological studies were used to provide an approximation of the changes in mortality and hospital admissions rates corresponding to changes in ozone levels from the 1990s to the 2050s. Because epidemiological studies differ in their estimated concentration-response functions, we calculated health impact estimates from multiple epidemiological studies. The concentration-response functions for a specific health endpoint can vary due to data availability, statistical methods, the communities' underlying health status, and other factors. In the future, the relationship between ambient pollution levels and health effects may change, for example due to changes in daily activity patterns and health care systems. Still, the present day relationships between ozone and health can provide a useful gauge of the health impacts that may occur with future changes in air quality. We choose epidemiological studies based on US cities (Bell et al. 2004, 2005; Levy et al. 2001; Medina-Ramón et al. 2006; Moolgavkar et al. 1997; Schwartz 1994, 1995, 2005; Sheppard 2003; Stieb et al. 2002, 2003; Thurston and Ito 2001).

3 Results

3.1 Ozone concentrations

Under the IPCC A2 climate scenario, the simulated daily 1- and 8-h maximum ozone levels during the summer are projected to rise 4.8 and 4.4 ppb, respectively, from the 1990s to the 2050s, averaged over the 50 cities. For comparison, Hogrefe et al. (2004a) reported an

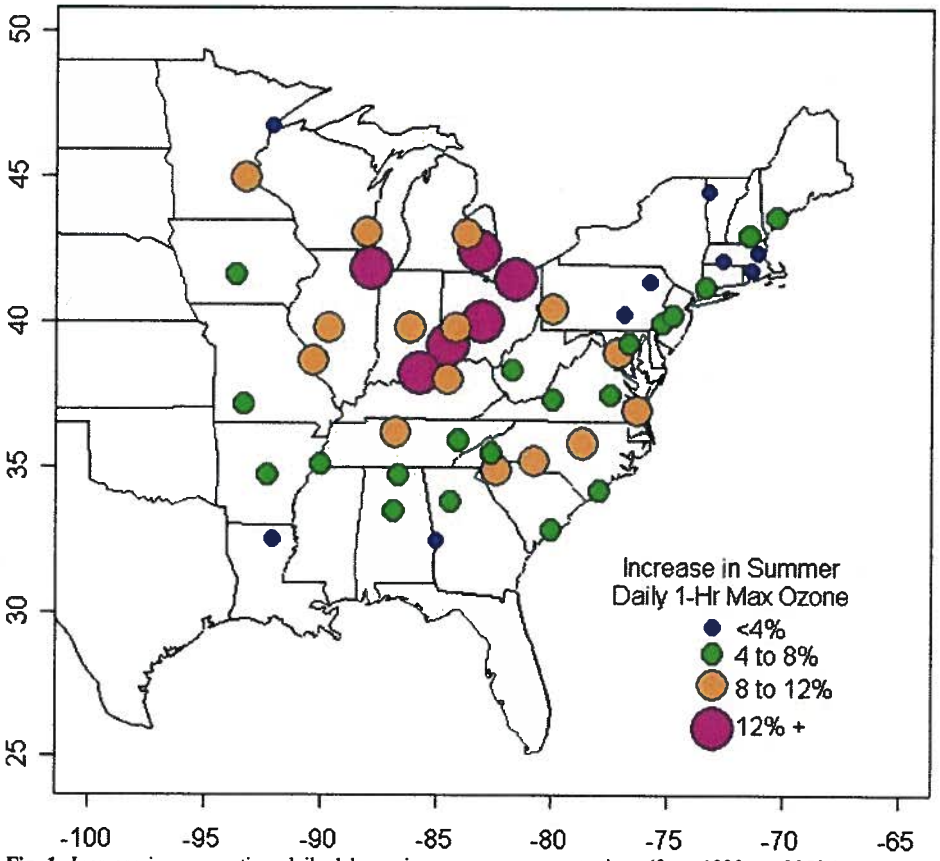

Fig. 1 Increase in summertime daily 1-h maximum ozone concentrations (from 1990s to 2050s)

increase of 4.2 ppb for summertime average 8-h daily maximum ozone concentrations averaged over 428 ozone monitors in the modeling domain for the same simulation.

The increases in ozone levels showed substantial spatial variation, as shown by Fig. 1. Table 2 provides the increase in ozone levels for several concentration metrics and shows the largest and smallest increase for any of the 50 cities. The future climate scenario caused higher daily 1-h maximum and daily 8-h maximum ozone concentrations for all cities. The daily average for all cities was also raised under the climate change scenario, except for one city that had basically the same daily average (0.01 ppb lower for the climate change scenario than the current emissions scenario).

Table 2 Changes in summer ozone concentrations comparing the projected future climate (2050s) to current climate (1990s), for 50 eastern US cities

	Average increase	Smallest increase[a]	Largest increase[a]
Daily average	2.9 ppb (6.4%)	−0.01 ppb (−0.02%)	6.4 ppb (13.1%)
Daily 1-h max	4.8 ppb (7.4%)	0.51 ppb (0.8%)	9.6 ppb (14.3%)
Daily 8-h max	4.4 ppb (7.2%)	0.45 ppb (0.8%)	9.0 ppb (13.7%)

[a] The smallest and largest city-specific effect represents the change averaged across all summers for any single city.

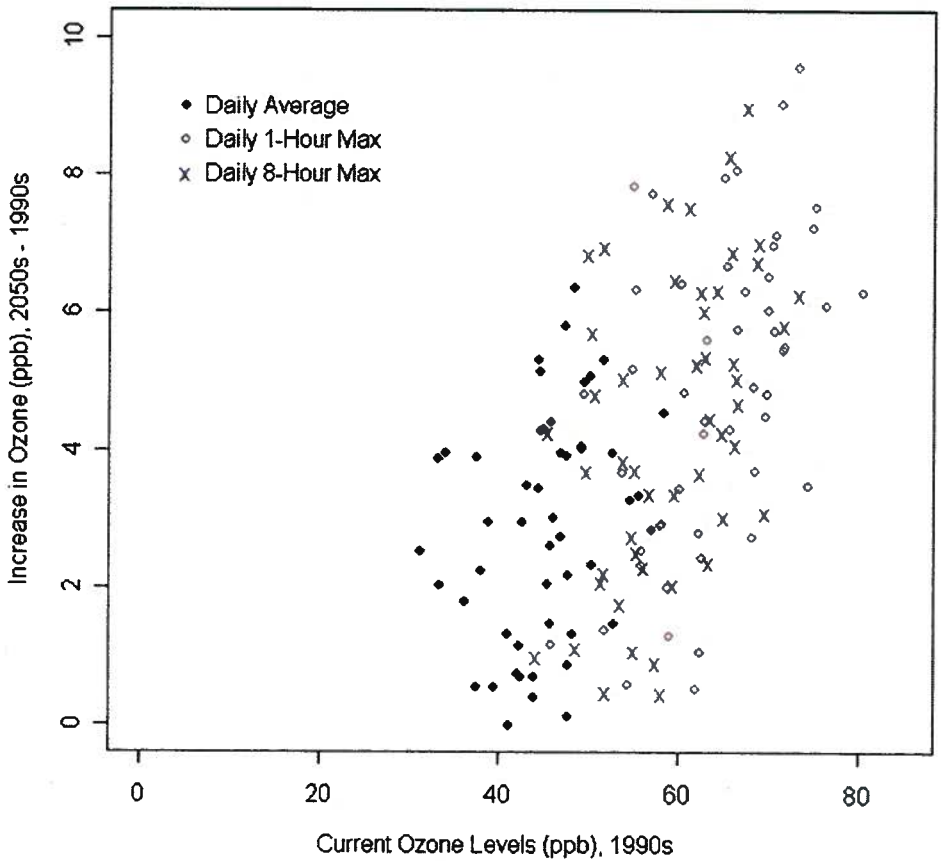

Fig. 2 Summer ozone concentrations under the current climate scenario (1990s) and increases under the future A2 climate scenario (2050s)

Cities that already experience elevated ozone levels under the current climatic conditions are predicted to exhibit the largest increases in ozone in the projected future climate scenario (Fig. 2). The higher increases in ozone for areas with present-day high pollution levels may result from these regions' correspondingly high emissions of ozone precursors, such as transportation and industrial pollution, as well as possibly biogenic emissions. The correlation between the increase in ozone and the baseline 1990s ozone levels is 0.55. The increase in ozone levels for each city was not purely caused by the increase in temperature for that city; other factors such as changes in biogenic emissions and changes in circulation patterns and mixing heights also played a role. This is demonstrated in Fig. 3, which shows the increase in the daily average ozone summer levels as a function of the increase in temperature. The correlation between the increase in summer temperature and the increase in ozone is 0.2.

3.2 Regulatory exceedances

We calculated the number of NAAQS exceedance days for each of the 10 summers for each city, using both the 1- and 8-h NAAQS standards, to estimate how the anticipated number of exceedance days would differ under the projected future climate scenario. All cities had

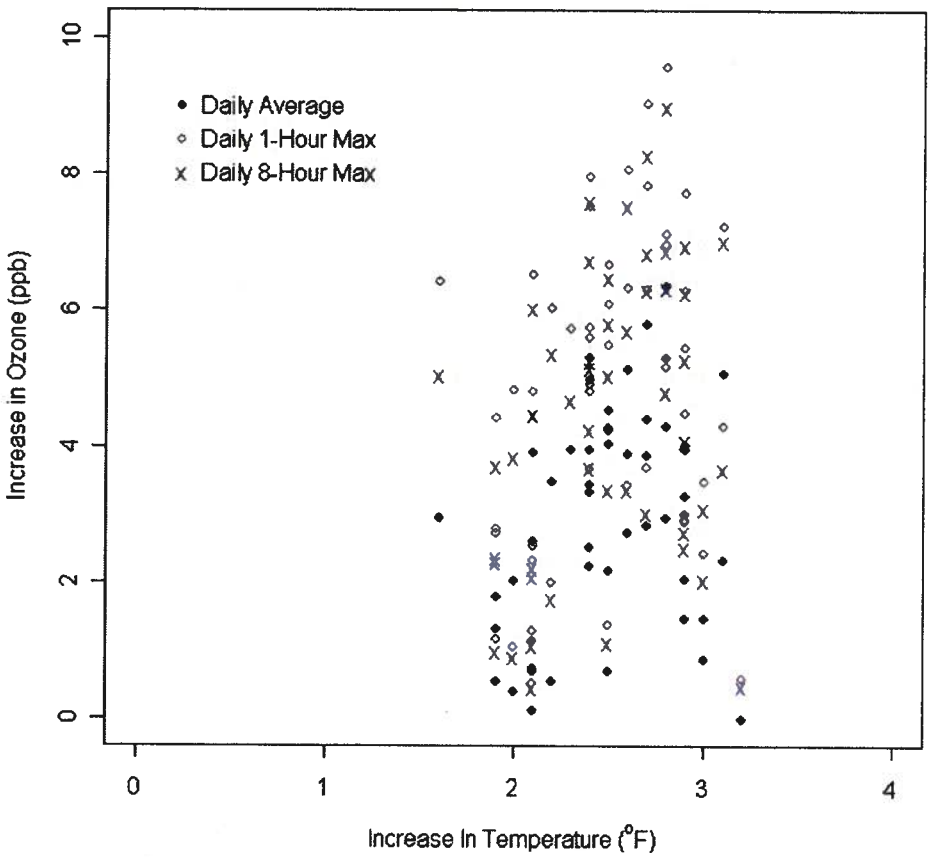

Fig. 3 Increases in temperature and summer ozone concentrations comparing the current climate scenario (1990s) to the future A2 climate scenario (2050s)

more or the same number of exceedance days under the projected future climate than under the current climate scenario for both regulatory standards. Figures 4 and 5 depict the average number of exceedance days per summer for each city for the 1- and 8-h ozone NAAQS, respectively. The blue horizontal bars represent the average number of exceedance days per summer for the current climate, whereas the red bars represent the additional number of exceedance days that would occur under the projected future climate scenario.

For both the present-day and projected future climate scenarios, all cities had the same number or more 8-h NAAQS exceedance days as 1-h NAAQS exceedance days. This is anticipated given that the 8-h standard is generally more stringent than the 1-h requirement (Bell and Ellis 2003). Cities with higher pre-existing ozone levels exhibited the largest increases in the number of exceedance days.

For the 1-h NAAQS, most cities (37) had no simulated 1-h NAAQS exceedance days under the current climate. However, because of model biases (Hogrefe et al. 2004b) the simulated current climate scenario ozone estimates do not represent actual ozone quality with respect to the NAAQS. For example, Greenville, SC, Nashville, and Roanoke are currently in non-attainment for the 1-h ozone standard although no 1-h exceedance days were estimated for the five 1990's summers (USEPA 2006). Sixteen of these 37 cities with

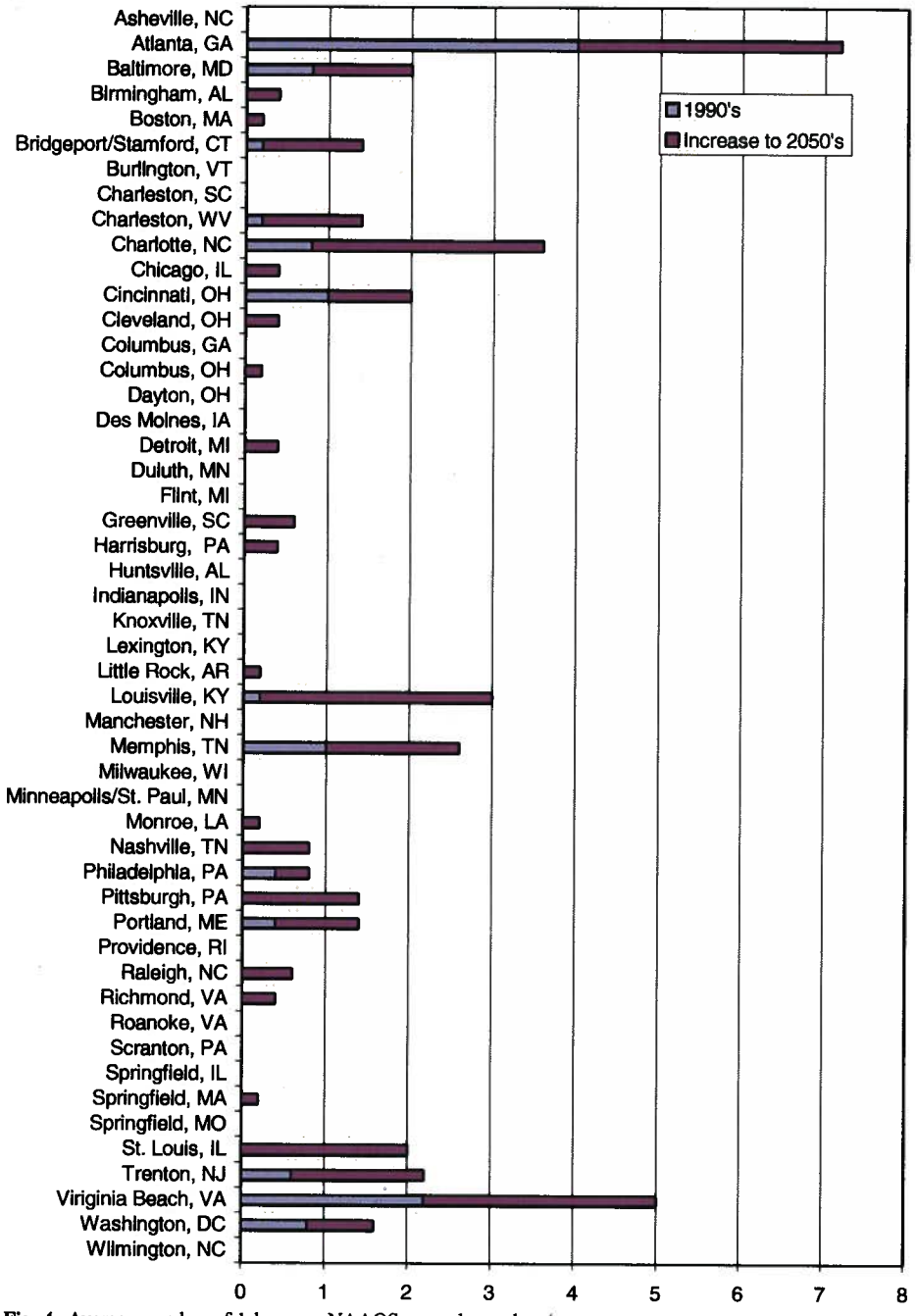

Fig. 4 Average number of 1-h ozone NAAQS exceedance days/summer

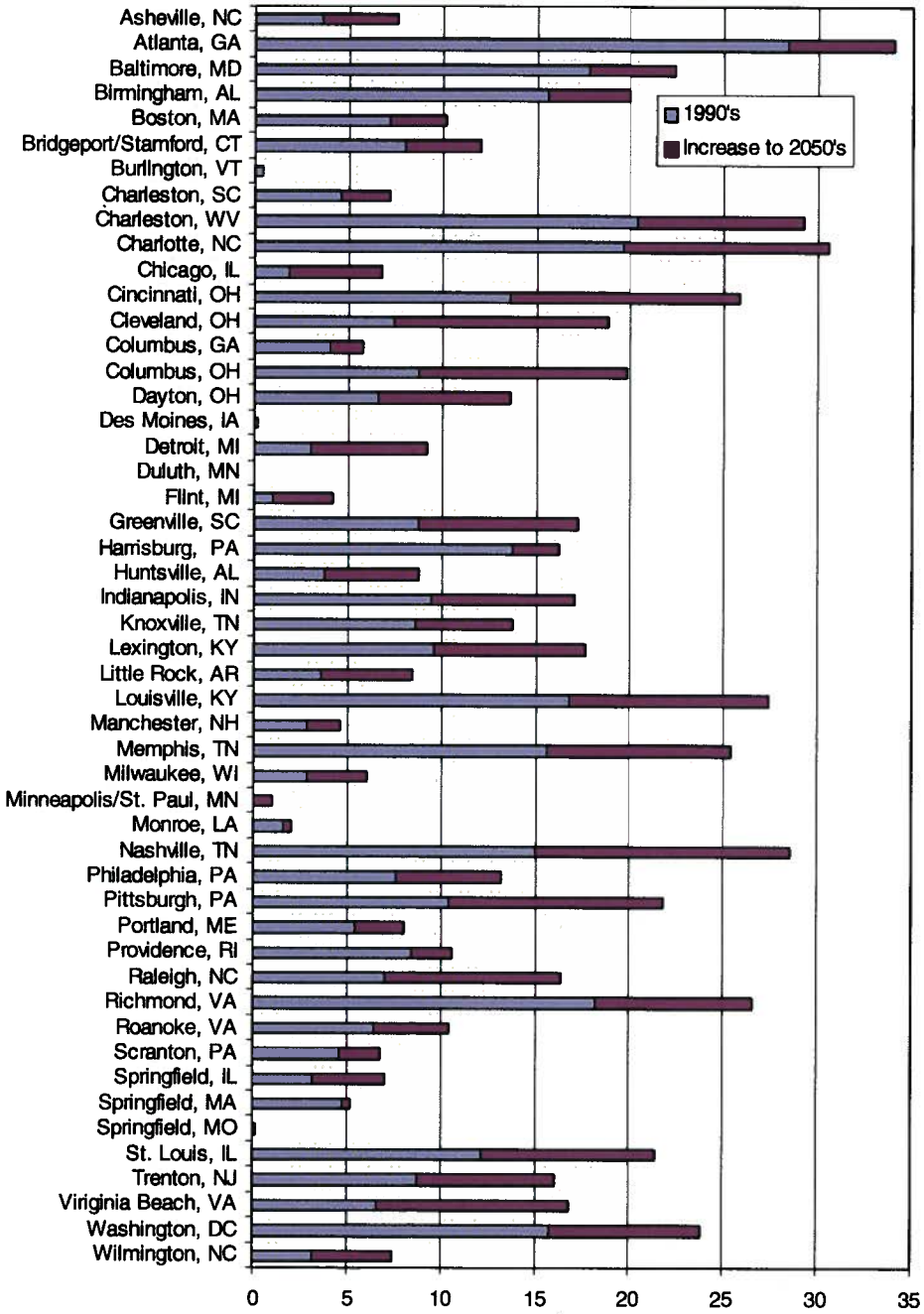

Fig. 5 Average number of 8-h ozone NAAQS exceedance days/summer

no simulated 1-h NAAQS exceedances in the current summers exceeded the 1-h requirement under projected future climatic conditions in the 2050s. On average across all the cities, there are an estimated 0.6 more exceedance days per summer in the 2050s than in the 1990s summers. For areas that exceeded the requirement for the 1990s summers, an average 1.7 more exceedance days/summer are predicted. The largest increase was 3.2 more 1-h exceedance days/summer for the city with the highest number of exceedance days/summer under the present day climate.

For the 8-h NAAQS, only two cities had no simulated 8-h exceedance days under the current climate, however one of these did exceed the standard using projected future climatic conditions. On average across all cities, there were 5.5 more 8-h NAAQS exceedance days/summer with the projected future climate than with the current climate, which is a 68% increase. In their analysis of simulated changes in the number of 8-h ozone NAAQS exceedances at the location of 428 O_3 monitors in the modeling domain, Hogrefe et al. (2004a) reported an increase of such exceedances by 65% from the 1990s to the 2050s.

3.3 Ozone action days

Figure 6 depicts the percent of summer days under each AQI ozone category for current conditions and the A2 projected future climate scenario, on average across the 50 cities. No city had maroon levels, the worst category, under either current or projected future conditions. Even under the current climate, 37% of the summer days in these 50 cities had an ozone AQI of yellow or worse, and 9% of the days had unhealthy conditions with an ozone AQI of orange or worse. Under the A2 climate change scenario for the 2050s, 47% of the days had yellow or worse ozone AQIs and 16% were at orange or worse categories, on average across the cities. The climate change scenario altered the distribution of AQI categories, with more days in each of the categories with adverse health effects (yellow, orange, red, and purple) and fewer days in the good ozone level category (green).

All cities had the same or more days with unhealthy ozone levels in the projected future climate. Several cities without red or higher AQI levels in the current scenario reach red

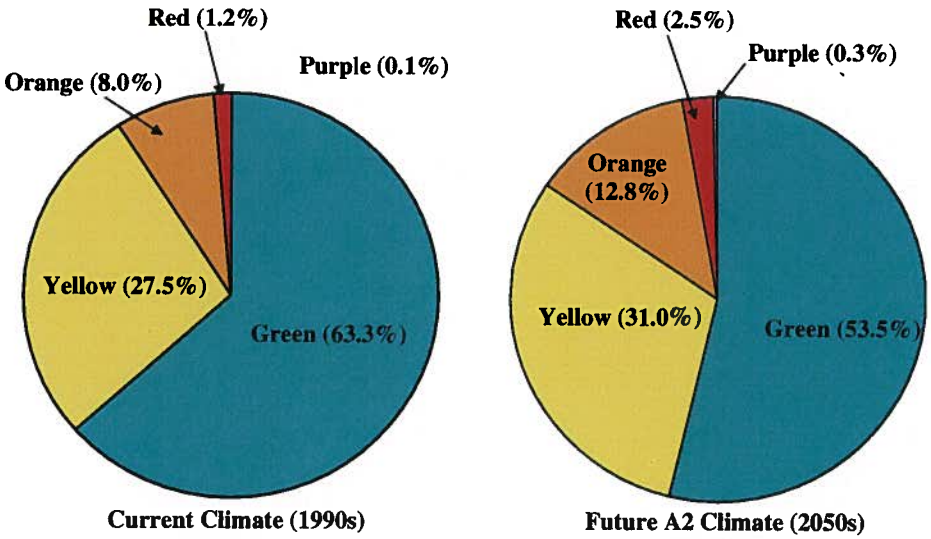

Fig. 6 Percent of summer days in each ozone air quality index category, on average across the 50 cities

Table 3 Percent increase (95% confidence intervals) from the 1990s to the 2050s in cause-specific hospital admissions

	Average across all cities	Largest city-specific effect[a]	Epidemiological study	Age category
COPD	0.8% (−0.2, 1.8)	1.8% (−0.4, 3.96)	[Moolgavkar et al. 1997]	≥65
	1.6% (0.4, 2.8)	3.5% (0.9, 6.27)	[Schwartz 1994]	≥65
	0.24% (0.07, 0.41)	0.49% (0.14, 0.85)	[Medina-Ramón et al. 2006]	≥65
Respiratory	0.8% to 2.1%	1.7% to 4.6%	[Schwartz 1995]	≥65
Asthma	2.1% (0.6, 3.6)	4.7% (1.4, 8.1)	[Sheppard 2003]	≤64

[a] The largest city-specific effect represents the largest change averaged across all summers for any single

levels with the projected future climate (Asheville, Chicago, Columbus, GA, Detroit, Flint, Little Rock, Raleigh, Springfield, IL, and Springfield, MA). Ten cities without purple AQIs in the current scenario have those levels under projected future climatic conditions (Boston, Harrisburg, Louisville, Nashville, Philadelphia, Pittsburgh, Portland, ME, Richmond, St. Louis, and Trenton).

3.4 Human health impacts

The increase in ozone concentrations resulting from climate change would be accompanied by a rise in the associated adverse health effects, such as premature death, hospital admissions and emergency department visits, exacerbation of asthma, and decreased lung function (Dockery and Pope 1994; Lippmann 1989, 1993; Thurston and Ito 1999; USEPA 2006). We estimated the change in selected health endpoints from elevated ozone levels caused by climate change using concentration-response relationships derived from epidemiological studies (Bell at al. 2004, 2005; Levy et al. 2001; Medina-Ramón et al. 2006; Moolgavkar et al. 1997; Schwartz 1994, 1995, 2005; Sheppard 2003; Stieb et al. 2002, 2003; Thurston and Ito 2001).

Table 4 Percent increase (95% confidence interval) from the 1990s to the 2050s in total mortality from elevated ozone levels in response to climate change

	Average across all cities	Largest city-specific effect[a]	Epidemiological study
Total	0.15% (0.08, 0.22)	0.33% (0.17, 0.48)	[Bell et al. 2004][b]
	0.11% (0.005, 0.21)	0.22% (0.01, 0.42)	[Schwartz 2005][c]
	0.24% (0.15, 0.32)	0.53% (0.34, 0.71)	[Bell et al. 2005]
	0.27% (0.16, 0.38)	0.63% (0.38, 0.88)	[Levy et al. 2001]
	0.21% (0.06, 0.37)	0.43% (0.12, 0.73)	[Stieb et al. 2002, 2003]
	0.26% (0.15, 0.37)	0.52% (0.30, 0.75)	[Thurston and Ito 2001]
Cardiovascular	0.31% (0.20, 0.42)	0.68% (0.43, 0.92)	[Bell et al. 2005]
Respiratory	0.12% (−0.14, 0.38)	0.27% (0.00, 0.85)	[Bell et al. 2005]
Cardiovascular and respiratory	0.18% (0.09, 0.28)	0.41% (0.20, 0.62)	[Bell et al. 2004][b]

[a] The largest city-specific effect represents the largest change averaged across all summers for any city.

[b] These results use concentration-response functions derived from a multi-city time-series study. Other results are from meta-analyses, except where specified.

[c] These results use concentration-response functions derived from a multi-city case-crossover study. Other results are from meta-analyses, except where specified.

Table 3 provides the percent increase in hospital admissions for persons aged 65 and over for chronic obstructive pulmonary disease (COPD) and respiratory causes, and for those aged 64 and younger for asthma-related causes. Results are provided for the average increase in cause-specific hospital admissions across all the cities, and the largest city-specific effect. The 95% confidence intervals reflect uncertainty in the concentration-response functions from the epidemiological studies, not uncertainty relating to the climate change predictions.

We estimated the percent increase in non-accidental mortality for all causes (total mortality) and for cardiovascular and respiratory mortality for these 50 cities based on elevated ozone levels from climate change using several epidemiological studies (Table 4). The actual number of deaths corresponding to these percent increases will depend on the true population and mortality rates in the 2050s. The percent increase in mortality shows some variation in relation to the concentration-response functions derived from various time-series studies. The epidemiological studies for mortality include meta-analysis studies (Bell et al. 2005; Levy et al. 2001; Stieb et al. 2002, 2003; Thurston and Ito 2001), a multi-city time-series study (Bell et al. 2004), and a multi-city case-crossover study (Schwartz 2005). Variation in the concentration-response functions for mortality may be due in part to publication bias in the meta-analysis studies.

4 Discussion

Our findings indicate that recently reported (Hogrefe et al. 2004a) potential increases in tropospheric ozone due to climate change would be accompanied by rises in the health outcomes associated with ozone for these 50 cities, including an increase in total, cardiovascular, and respiratory mortality; hospital admissions for asthma; and hospital admissions for COPD and respiratory causes for older populations. Even at current levels, much of the US is not in compliance with the health-based regulatory requirements for ozone.

The mortality and hospital admission impacts represent only a fraction of the health toll that would be caused by elevations in tropospheric ozone levels. For mortality, the concentration-response functions account for acute exposure and thereby could underestimate the total effect of ozone on mortality through long-term exposure. Other health endpoints include respiratory symptoms such as shortness of breath, restricted activity days, school absenteeism, emergency room visits, asthma attacks, and lung inflammation, in addition to ecological and welfare effects such as material damage (USEPA 1999).

The results presented here reflect the impact of an altered climate on ozone and human health with anthropogenic emissions held constant, however a full portrayal of the possibilities for future ozone levels would include an array of scenarios for anthropogenic emissions of ozone precursors as well as a variety of climate change scenarios based on GHG emissions. Several policies are underway on national and local levels to lower ozone concentrations. Examples are EPA's Clean Air Interstate Rule (CAIR) and city-level actions in transportation planning. Results revealed that the largest increases in ozone levels are predicted to occur in cities that already have high pollution levels, however it may be these cities that work more diligently to lower emissions of ozone precursors in the future. The results presented here indicate that such locations may have to increase their emission control efforts to counteract the adverse impact of climate change on ozone concentrations.

On a larger scale, we could envision different results from various IPCC scenarios. For instance, the A1 scenario has rapid economic expansion, a low population rise, and increased introduction and use of energy efficient technologies. The A2 scenario applied

here has more regionally oriented economic development with slower per capita economic expansion and technological advancement (Nakicenovic and Swart 2000). Due to the range of options for population growth, energy technologies, and policies on local, regional, and global scales, the prediction of ozone levels in the 2050s is quite difficult (Kinney et al. 2005; Schwartz et al. 2005]. In this work, we aim to demonstrate the application of a modeling system to a specific scenario in order to evaluate future ozone levels under conditions of climate change, however various emissions scenarios could result in different ozone levels. Future work will examine a broader range of scenarios.

Another caveat of this work is uncertainty in the various stages of the modeling system. For example, cloud cover may be altered in a future climate, which could affect ozone formation. A recent study estimated changes in low-level cloud cover over the continental US leading to elevated ozone formation for a future climate scenario (Murazaki and Hess 2006). However, the authors noted that the nature of clouds under a future climate scenario is highly uncertain (Murazaki and Hess 2006; Stocker 2001).

The adverse health impacts from ozone in relation to climate change, as well as the many other potential health consequences of climate change such as altered distribution patterns for vector-borne diseases, highlight the need for diligence regarding policies that control greenhouse gas emissions and that improve regional air quality. Such policies can have short-term local benefits to air quality, and thereby human health, in addition to their effects on long-term climate change and health. Thus, it would be worthwhile to incorporate climate concerns into air quality planning, and similarly to consider the short-term health benefits of climate-change mitigation and adaptation policies.

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Rising CO_2 and pollen production of common ragweed (*Ambrosia artemisiifolia*), a known allergy-inducing species: implications for public health

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Abstract. Although environmental factors such as precipitation and temperature are recognized as influencing pollen production, the impact of rising atmospheric carbon dioxide concentration ($[CO_2]$) on the potential growth and pollen production of hay-fever-inducing plants is unknown. Here we present measurements of growth and pollen production of common ragweed (*Ambrosia artemisiifolia* L.) from pre-industrial $[CO_2]$ (280 μmol mol^{-1}) to current concentrations (370 μmol mol^{-1}) to a projected 21st century concentration (600 μmol mol^{-1}). We found that exposure to current and elevated $[CO_2]$ increased ragweed pollen production by 131 and 320%, respectively, compared to plants grown at pre-industrial $[CO_2]$. The observed stimulations of pollen production from the pre-industrial $[CO_2]$ were due to an increase in the number (at 370 μmol mol^{-1}) and number and size (at 600 μmol mol^{-1}) of floral spikes. Overall, floral weight as a percentage of total plant weight decreased (from 21% to 13%), while investment in pollen increased (from 3.6 to 6%) between 280 and 600 μmol mol^{-1} CO_2. Our results suggest that the continuing increase in atmospheric $[CO_2]$ could directly influence public health by stimulating the growth and pollen production of allergy-inducing species such as ragweed.

Keywords: allergens, elevated carbon dioxide, photosynthesis, pollen, relative growth rate.

Introduction

Since the start of the Industrial Revolution, atmospheric $[CO_2]$ has risen from ~280 μmol mol^{-1} to ~370 μmol mol^{-1} (Houghton *et al.* 1996). This rise in atmospheric $[CO_2]$ has not been linear. Approximately two-thirds of the observed increase in $[CO_2]$ has occurred since the 1950s (Keeling and Whorf 1994). Although the projected rate of future atmospheric $[CO_2]$ increase varies by model, it is generally acknowledged that atmospheric $[CO_2]$ should reach 600 μmol mol^{-1} sometime during the 21st century (Houghton *et al.* 1996).

Because CO_2 supplies the carbon for all terrestrial biology, research efforts have focused on determining the impact of rising atmospheric $[CO_2]$ on the growth and reproduction of native species and crops (e.g. Kimball *et al.* 1993; Poorter 1993; Curtis and Wang 1998). Reproduction is an especially important parameter since it affects both ecological fitness for native species, and economic production for crops.

The impact of $[CO_2]$ on allocation of resources to flowering and changes in reproductive phenology appears to be highly species-specific (Ackerly and Bazzaz 1995). Enhanced $[CO_2]$ has resulted in earlier flowering (Lawlor and Mitchell 1991) and increased flower and fruit number for a number of agronomic plants (Deng and Woodward 1998). In contrast, flowering of some native species has been unaffected or delayed with increased $[CO_2]$ (Garbutt and Bazzaz 1984; Reekie *et al.* 1997), but this is by no means a universal response (e.g. *Datura stramonium*, Garbutt and Bazzaz 1984).

While specific attention has been given to aspects of reproductive biology which could influence seed development and yield, almost nothing is known concerning the impact of $[CO_2]$ on pollen production *per se*. Aside from the obvious consequences for fertilization, fecundity and ecological fitness, $[CO_2]$-induced changes in pollen production could have a direct impact on atmospheric pollen concentration, with subsequent effects on human allergic disease and public health. For example, in a recent survey among the general population in the US, approximately 70% of respondents indicated pollen as the principal agent producing symptoms of allergies, with ragweed pollen cited as the individual plant species eliciting the greatest response (Meggs *et al.* 1996). In addition to ragweed, other weedy species (e.g. lambsquarters, *Chenopodium album* L.) and pigweed (*Amaranthus retroflexus* L.) as well as native trees

Abbreviations used: A, rate of CO_2 assimilation; C_a, ambient CO_2 concentration; C_i, internal CO_2 concentration; DAS, days after sowing; PPFD, photosynthetic photon flux density; RGR, relative growth rate.

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(e.g. *Quercus* and *Acer* spp.) and grasses (e.g. *Setaria*) are recognized as influencing seasonal allergies through pollen production (Gergen and Turkeltaub 1992; Emberlin 1994).

In the current experiment, our principal objective was to test whether the increase in atmospheric [CO_2] since the Industrial Revolution, and projected future increases in [CO_2], may alter growth and pollen production of known hay-fever-inducing plants using common ragweed (*Ambrosia artemisiifolia* L.) as a model species. Pollen production of ragweed is generally acknowledged to be a major source of air-borne allergens and a public health concern in North America. In general, ragweed production peaks between late August and November in North America, and is the principal pollen associated with fall allergies in the US (Frenz *et al.* 1995; Meggs *et al.* 1996). CO_2-induced changes in the life cycle and pollen-producing capacity of aero-allergen plants such as ragweed could have significant implications for public health.

Materials and methods

Experiments were conducted using a controlled environment chamber located at the Climate Stress Laboratory, USDA-ARS, Beltsville, MD, USA. An environmental chamber was used rather than field chambers or Free-Air CO_2 Exchange (FACE), in order to maintain constant pre-industrial [CO_2] at a given temperature and consistent light and humidity for 24-h periods.

[CO_2] was controlled by flushing the chamber with CO_2-free air using a Ballston 75-60 type CO_2 scrubber (Ballston Filter Products, Lexington, MA, USA), then re-injecting CO_2 to the desired [CO_2]. Injection of CO_2 was controlled by an infrared gas analyser (WMA-2, PP systems, Haverhill, MA, USA) in absolute mode that sampled chamber air continuously. The set points for [CO_2] control were 280 (pre-industrial), 370 (current) and 600 (future) μmol mol^{-1} CO_2. Actual CO_2 concentrations determined at 10-min intervals over 24 h for each [CO_2] treatment were 281.5 ± 23.4, 374 ± 14.1 and 603 ± 12.9 μmol mol^{-1}, respectively. In all chambers, plants received 14 h of 1.0 mmol m^{-2} s^{-1} photosynthetic photon flux density (PPFD) from a mixture of high-pressure sodium and metal halide lamps for the first 35 days after sowing (DAS). After 35 DAS, PPFD was altered to 12 h of 1.0 mmol m^{-2} s^{-1} PPFD to induce flowering. Day/night temperature was 28/22°C and average daily humidity exceeded 60%. Temperature, [CO_2] and relative humidity were monitored and recorded at 1-min intervals by an EGC network data logger (EGC Corp., Chagrin Falls, OH, USA) in conjunction with a PC.

Seeds of common ragweed (*Ambrosia artemisiifolia* L.) were broadcast in pots of different sizes ranging from 10 to 30 cm in diameter (1.8–21.2 L in volume). Smaller pots were elevated so that the height of the plants was uniform. Seed was obtained from the Valley Seed Company (Fresno, CA, USA). Plants in all pots were thinned to one plant per pot within 48 h after emergence. Pots were filled with vermiculite and watered daily to dripping point with a complete nutrient solution containing 13.5 mM nitrogen (Robinson 1984). For each experiment, 32 pots were assigned to a given [CO_2], with pots arranged to avoid mutual shading.

To determine potential changes in photosynthesis as a function of the growth [CO_2], single leaf photosynthesis (A, the rate of CO_2 assimilation) was determined as a function of short-term changes in internal [CO_2] (C_i) twice during the vegetative growth at each [CO_2] treatment. Assimilation was determined on the uppermost, fully expanded leaf for four plants of each [CO_2] between 30 and 40 DAS using a differential infrared CO_2 analyser (model 6252, Li-Cor Corp., Lincoln, NE, USA) in an open configuration attached to two single-leaf cuvettes. Temperature, humidity and [CO_2] were set to approximate values maintained in the growth chamber. The gas stream was humidified by bubbling through a temperature-controlled water bath to obtain a given dew point, and humidity was monitored with a dew point hygrometer (Hygro M-1, General Eastern Corp., Cambridge, MA, USA). Mass flow controllers were used to mix dry CO_2-free air with 100% CO_2 to obtain a desired [CO_2] within a cuvette. Supplemental lighting was supplied by a 150-W cool-beam floodlight (GE Corp., Cleveland, OH, USA) attached to a variable transformer to obtain a desired PPFD.

Photosynthesis was determined initially as the CO_2 assimilation rate at the growth [CO_2] (C_a) at the growth PPFD (1.0 mmol m^{-2} s^{-1}), then re-measured at saturating light intensity (1.6 mmol m^{-2} s^{-1}). C_a was then reduced to 90 μmol mol^{-1} and increased in steps to 180, 360, 720, 1080 and 1450 μmol mol^{-1}. Sufficient time (usually 20–30 min) was given after C_a was changed, to allow equilibration. At the end of the measurement, leaf laminae contained within a cuvette were cut, and leaf area determined with a leaf area meter (Li-Cor Corp.).

For each [CO_2] treatment, six plants were harvested at 21, 25, 29 and 35 DAS and again at seed maturity. Seed maturity was defined as occurring when seed set exceeded 90%. At each harvest, all plants for a given [CO_2] treatment were cut at ground level and separated into leaf laminae, stems (including petioles) and roots. Smaller-volume pots were harvested first to avoid root-binding effects. Total leaf area was determined photometrically as described previously. Dry weights were obtained separately for leaves, stems and roots. Material was dried at 65°C for a minimum of 72 h or until dry weight was constant, and then weighed. For the maturity harvest, leaf area was estimated based on the regression analysis between leaf area and weight obtained from previous harvests ($R^2 = 0.99$).

Before pollination, 10 terminal staminate floral spikes (catkins) were selected on each of five plants from each [CO_2] treatment, and labeled. A 5 × 25 cm polyethylene bag was placed over each spike to collect pollen. Each bag had a 2.5-cm slit cut approximately 2 cm from the bottom of the bag, into which the floral spike was placed with the peduncle of the raceme located at the bottom of the slit. After placement of the bag, the slit was taped so the floral spike was inside the bag with at least 5 cm of space from the top of the open bag. Tops of bags were left open for air circulation and ventilation. Floral heads were tapped gently each day, and pollen was allowed to fall to the bottom of the bag. After flowers were dehiscent and heads had completed pollen production, each bagged floral spike was cut immediately below the first flower and the floral structure was removed from the bag after tapping any residual pollen. Each spike was measured for length along with fresh and dry weights. Total pollen for a given spike was calculated by subtracting the initial bag weight from the bag and pollen weight. At maturity, for each plant from each [CO_2] treatment, the total number of floral spikes was recorded, spikes were harvested and the dry weight (without pollen) recorded. The ratio of pollen collected to dry weight of the floral structure resulted in a consistent ratio that was used to estimate pollen production per plant.

Because of potential differences in microclimate between chambers, the same growth chamber was used for all three CO_2 levels. Adjustments to PPFD, humidity and temperature control were made prior to the start of each [CO_2] treatment to maintain consistency in microclimate. In addition, the entire experiment (i.e. all three [CO_2] treatments) was repeated. A two-way ANOVA (SuperANOVA, Abacus Concepts, Berkeley, CA, USA) was used to test for differences between the two runs and between treatments. Because no significant run effect was detected, treatment effects were compared with a one-way analysis of variance on the combined data. Final biomass for a given [CO_2] treatment differed by <10% between runs. Three separate post-hoc tests (Student–Newman–Keuls, Duncan New Multiple Range and Fisher's

protected LSD) determined differences at the 0.05 significance level as a function of [CO_2] treatment.

Results

Pollen production increased significantly with rising [CO_2]. The observed increase was 132% from pre-industrial to current CO_2 levels, and ~90% from current to future CO_2 levels of 600 μmol mol^{-1} (Fig. 1). Sensitivity of pollen production to increasing [CO_2] was greater from pre-industrial to current CO_2 levels (0.7 g of pollen per 10 μmol mol^{-1} increase in [CO_2]), diminishing as CO_2 increased to 600 μmol mol^{-1} (0.4 g of pollen per 10 μmol mol^{-1} increase in [CO_2]). Floral spike number did not change from pre-industrial to current [CO_2], but pollen production per spikelet increased significantly (Fig. 1). From 370 to 600 μmol mol^{-1} CO_2, no further change in pollen production per spikelet was noted, but the number of floral spikes approximately doubled (Fig. 1). Analysis of the diameter of 200 individual pollen grains for each [CO_2] using a SEM (15 kV, ×1.5k) indicated no change in average pollen size (data not shown).

Small (non-significant) changes in total biomass (i.e. roots, stems and leaves) were observed by 21 DAS, and significant differences were observed by 29 DAS (Fig. 2). Significant differences in relative growth rate (RGR) also occurred by 29 DAS among [CO_2] treatments (0.174, 0.209 and 0.220 g g^{-1} day^{-1} for the 280, 370 and 600 μmol mol^{-1} [CO_2] treatments, respectively).

At seed maturity, total plant biomass was directly proportional to [CO_2]. From pre-industrial to current atmospheric [CO_2], leaf weight and stem weight increased by 36 and 49%, respectively, with no significant change in root or floral weight (Table 1). Leaf area, however, almost doubled in size for this same increase in [CO_2] (Table 1). At 600 μmol mol^{-1}, significant increases in all growth parameters at maturity were observed relative to the 370 and 280 μmol mol^{-1} treatments. The largest relative increase was observed for root weight which increased ~ 4-fold from 280 to 600 μmol mol^{-1} [CO_2]. No significant changes in stem to root ratio or specific leaf area occurred among [CO_2] treatments (data not shown).

Leaf photosynthesis, measured at the growth [CO_2], increased significantly with [CO_2], rising 170 and 250% from pre-industrial to current and future [CO_2], respectively, and 30% from current to future [CO_2] (Table 2). Values of assimilation at a measurement [CO_2] of 280 μmol mol^{-1} did not differ among [CO_2] treatments. However, at higher measurement CO_2 levels, leaves grown at 280 μmol mol^{-1} [CO_2] had significantly lower photosynthetic rates — an indication of down-regulation (Table 2). Acclimation was not observed between the 370 and 600 μmol mol^{-1} [CO_2] treatments. Analysis of the response of assimilation to internal CO_2 indicated no significant change in the initial slope of the response curve as a function of [CO_2] treatment. However, the internal CO_2 concentration where leaf assimilation is equal to zero (Γ^*, the CO_2 compensation point), and the maximum observed assimilation rate was lower for the 280 than the 370 and 600 μmol mol^{-1} treatments (data not shown).

Discussion

For many years, both botanists and health workers have been interested in those climatic and/or meteorological factors that influence atmospheric pollen concentration (e.g. Gregory 1973; Buck and Levetin 1982). Abiotic factors that influence pollen productivity such as rainfall, temperature and light also determine pollen amounts and severity of

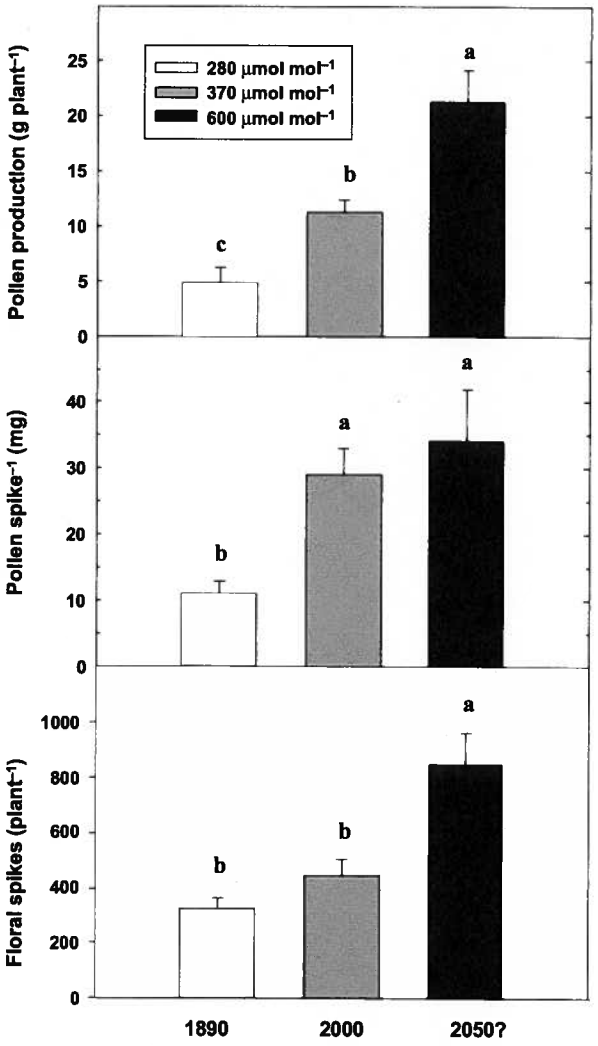

Fig. 1. Pollen production, pollen per floral spike and number of floral spikes in common ragweed grown at pre-industrial CO_2 concentrations (280 μmol mol^{-1}), current concentrations (370 μmol mol^{-1}) and a projected 21st century concentration (600 μmol mol^{-1}). Bars are ± s.e. Student–Newman–Keuls was used to determine differences among the [CO_2] treatments at the 0.05 significance level (a, b or c).

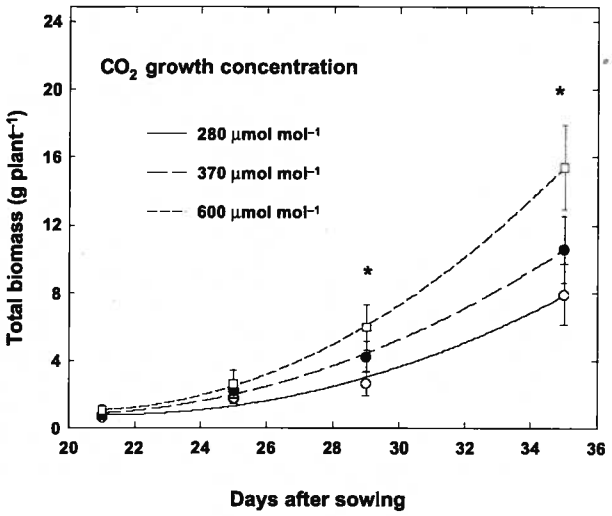

Fig. 2. Change in total plant biomass (g plant^{-1}) as a function of days after sowing (DAS) for ragweed grown at pre-industrial, current and future atmospheric [CO_2]. Bars are ± s.e. Relative growth rate (RGR) was determined between 21 and 29 DAS. No further change in RGR as a function of [CO_2] was observed after 35 DAS. * indicates a significant increase in total biomass relative to the 280 μmol mol^{-1} [CO_2] control.

allergies among susceptible populations during a given allergy season. Consequently, there has been a great deal of interest in modeling changes in pollen type and abundance associated with global climate change (Emberlin 1994). However, less is known concerning the direct stimulation of growth and pollen production of allergy-inducing species by rising [CO_2], one of the principal 'greenhouse' gases.

The observed stimulation in growth and photosynthesis observed here for ragweed is consistent with results seen elsewhere for C_3 species grown with enhanced [CO_2] (Kimball *et al.* 1993). Increasing the [CO_2] to 600 μmol mol^{-1} increased ragweed RGR within 4 weeks following emergence, and stimulated biomass at maturity almost 3-fold above pre-industrial [CO_2] values. Final vegetative biomass values obtained in the current experiment at ambient [CO_2] are consistent with those observed for single ragweed plants in abandoned agricultural fields at maturity (D. Patterson, pers. comm.). In the current study, both leaf area and weight were particularly sensitive to [CO_2]. The continued stimulation of single-leaf photosynthesis (at least through anthesis) and the observed increase in leaf area have obvious implications for maintaining a continued stimulation of photosynthesis and growth at the whole plant level with future CO_2 levels.

If growth of ragweed is indeed stimulated by increasing [CO_2], how does this alter subsequent reproductive effort? Floral spikes of ragweed contain both staminate and pistillate flowers. Pollen is wind-directed from numerous staminate flowers to pistillate heads, which are fewer in number and occur at the base of the floral spike (Bianchi *et al.* 1959). Because wind is the primary means of transport, large amounts of pollen are necessary to achieve seed set. Increasing vegetative growth provides both a structural platform for floral production and the carbon assimilate needed to produce flowers. In the current experiment, floral weight increased 70%, but floral weight as a percentage of total plant weight decreased (from 21% to 13%) from 280 to 600 μmol mol^{-1} CO_2. However, investment in pollen increased (from 3.6 to 6%) from 280 to 600 μmol mol^{-1} CO_2.

Because of the role of ragweed pollen in inducing allergies, the reproductive response of ragweed to rising atmospheric [CO_2] is of obvious interest. In the current study, the response of ragweed pollen production to rising [CO_2] was 2-fold. Increasing [CO_2] from pre-industrial to current levels increased the amount of pollen produced by an individual floral spike. As [CO_2] increased further to 600 μmol mol^{-1}, no additional increase in pollen per floral spike was observed, but the number of floral spikes rose significantly. The net result was an approximate doubling of pollen production capacity from pre-industrial to present day [CO_2] and a further doubling to a projected [CO_2] of 600 μmol mol^{-1}.

Interestingly, interpolation of the potential pollen response of ragweed to [CO_2] from the 1950s (~315 μmol mol^{-1}) to current levels shows a percentage

Table 1. Changes in measured (leaf area, leaf, stem and root dry weights) vegetative parameters at maturity for common ragweed (*Ambrosia artemisiifolia* L.) grown at pre-industrial, current and future levels of atmospheric [CO_2]

Different letters within a column indicate statistical differences between [CO_2] treatments at the 0.05 level according to Student–Newman–Keuls. Data are given on a per-plant basis

[CO_2] (μmol mol^{-1})	Area (m^2)	Weight (g)				Total weight (g)
		Leaf	Stem	Root	Floral	
280	1.15 c	65.1 c	30.7 c	11.3 b	28.9 b	135.1 c
370	2.17 b	88.7 b	45.7 b	13.5 b	35.7 b	183.6 b
600	3.41 a	178.9 a	97.1 a	50.3 a	49.2 a	372.4 a

Table 2. Photosynthesis (as CO_2 assimilation rate, μmol CO_2 m^{-2} s^{-1}) for ragweed (*Ambrosia artemisiifolia* L.) grown and measured at pre-industrial, current and future atmospheric [CO_2]

Different letters within a column indicate statistical differences between [CO_2] treatments at the 0.05 level according to Student–Newman–Keuls. Additional details are given in 'Materials and methods'

Growth [CO_2] (μmol mol^{-1})	Measurement [CO_2] (μmol mol^{-1})		
	280	370	600
280	15.1	23.3 b	33.1 b
370	19.6	40.7 a	53.0 a
600	14.8	35.6 a	52.9 a

increase in ragweed pollen consistent with the recent reported percentage increase in allergies and allergy-induced asthma among the general population (Platt-Mills and Carter 1997; Woolcock and Peat 1997). However, has the actual amount of ragweed pollen in the environment increased within the last 40 years? Because the rise in atmospheric [CO_2] has been so rapid, traditional ^{14}C dating techniques to determine *in situ* increases in pollen production since the mid-1950s are not applicable. The earliest pollen studies are based on some 13 000 gravity slide samples from 22 American cities summarized from 1916 to 1928 (Durham 1929). Unfortunately, direct comparisons between gravimetric and volumetric devices (e.g. Rotorod sampler) are difficult to perform. Differences in pollen recovery cannot be quantified, even roughly (see Frenz 1999*a*). Even if different sampling techniques were comparable, changes in land use and nitrogen deposition in industrial areas could not be separated from any direct atmospheric CO_2 effect. Current regional, on-site estimates of ragweed pollen production do not date back more than a few years (Frenz *et al.* 1995, Frenz 1999*b*), although it is hoped that these data could be used to verify potential increases in ragweed pollen with future increases in atmospheric [CO_2].

Will similar increases in pollen with enhanced [CO_2] be observed for other known allergy-inducing species? Projected increases in [CO_2] have been shown to stimulate the photosynthesis and growth of C_3 species such as lambsquarters (Carlson and Bazzaz 1982) and oak (Bunce 1992), as well as some C_4 species such as pigweed (Tremmel and Patterson 1993) and foxtail (Ziska and Bunce 1997). However, the reproductive response to [CO_2] cannot always be elucidated from observed increases in vegetative biomass (Jablonski 1997).

Critics of the role of CO_2 in climate change correctly point out that rising [CO_2] could result in a lush plant environment (Idso and Idso 1994). However, it should also be emphasized that the rise in [CO_2] is indiscriminatory with respect to the stimulation of both useful and noxious plant species. Furthermore, elimination of noxious weedy species by chemical means cannot always be assumed as atmospheric [CO_2] increases (Ziska *et al.* 1999). Consequently, the role of rising atmospheric [CO_2] with respect to distribution, growth and pollen production of weeds impacting human health should be of growing concern.

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CLIMATE CHANGE AND PUBLIC HEALTH IN CALIFORNIA

A Report From:
California Climate Change Center

Prepared By:
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California Air Resources Board

FINAL PAPER

DISCLAIMER

California Environmental Protection Agency
AIR RESOURCES BOARD

Arnold Schwarzenegger, *Governor*

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Preface

The California Energy Commission's Public Interest Energy Research (PIER) Program supports public interest energy research and development that will help improve the quality of life in California by bringing environmentally safe, affordable, and reliable energy services and products to the marketplace.

The PIER Program conducts public interest research, development, and demonstration (RD&D) projects to benefit California's electricity and natural gas ratepayers. The PIER Program strives to conduct the most promising public interest energy research by partnering with RD&D entities, including individuals, businesses, utilities, and public or private research institutions.

PIER funding efforts focus on the following RD&D program areas:

- Buildings End-Use Energy Efficiency
- Energy-Related Environmental Research
- Energy Systems Integration
- Environmentally Preferred Advanced Generation
- Industrial/Agricultural/Water End-Use Energy Efficiency
- Renewable Energy Technologies
- Transportation

In 2003, the California Energy Commission's PIER Program established the **California Climate Change Center** to document climate change research relevant to the states. This center is a virtual organization with core research activities at Scripps Institution of Oceanography and the University of California, Berkeley, complemented by efforts at other research institutions. Priority research areas defined in PIER's five-year Climate Change Research Plan are: monitoring, analysis, and modeling of climate; analysis of options to reduce greenhouse gas emissions; assessment of physical impacts and of adaptation strategies; and analysis of the economic consequences of both climate change impacts and the efforts designed to reduce emissions.

The California Climate Change Center Report Series details ongoing center-sponsored research. As interim project results, the information contained in these reports may change; authors should be contacted for the most recent project results. By providing ready access to this timely research, the center seeks to inform the public and expand dissemination of climate change information, thereby leveraging collaborative efforts and increasing the benefits of this research to California's citizens, environment, and economy.

For more information on the PIER Program, please visit the Energy Commission's website www.energy.ca.gov/pier/ or contract the Energy Commission at (916) 654-5164.

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Abstract

Population health is affected by a complex integration of many factors, including biological, ecological, social, and geographical inputs. Review of the literature on the public health consequences of climate change leads to the conclusion that the outcomes most likely to occur in California include death and illness related to temperature, air pollution, vector- and water-borne diseases, and wildfires. The population groups most at risk include the elderly, those with chronic heart or lung disease, children, people with mental illnesses or addictions, and the socially or economically disadvantaged. There are a number of actions and mitigations that can be adopted on the state-wide, local, and personal level that can reduce or prevent adverse health outcomes related to climate change. These actions include development of heat emergency action plans, continuing efforts to attain the health-based ambient air quality standards, strengthening surveillance for infectious diseases, expanding vector control programs, and review and modernization (as necessary) of water and sewage treatment facilities. It will also be important to expand public education on the health risks associated with climate change, including effective actions the individuals, caregivers, communities and health care providers can take to minimize personal risk.

Keywords: Climate change and public health; climate change and infectious diseases; heat mortality; air pollution and climate change; wildfires and public health

1.0 Introduction

The scientific community is in substantial agreement that the earth is warming, and that climatic patterns are changing, both worldwide and in California (IPCC 2007). Scenario analyses suggest that by the end of the twenty-first century temperatures in California could rise from 2°F to 9°F (1°C to 5°C), depending on the emissions scenario and general circulation model employed for the analysis (Cayan et al. 2009).

In June 2005 Governor Arnold Schwarzenegger issued Executive Order S-3-05, which set greenhouse gas emission reduction targets for California. This Executive Order also directed the Secretary of the California Environmental Protection Agency (CalEPA) to report to the governor and the state legislature by January 2006 (and biannually thereafter) on the impacts of global warming on California, including effects on public health.

Population health is affected by a complex integration of many factors, including biological, ecological, social, political, and geographical inputs. Because of this, the distribution of adverse public health impacts related to climate change is not expected to be uniform worldwide (Gamble et al. 2008), and not all potential public health impacts are expected to be significant concerns for the United States or California, compared to other regions of the world. The impacts identified as most likely to be concerns for public health in California are mortality and morbidity related to temperature, although adverse impacts related to air pollution, vector- and water-borne diseases, and wildfires are also of concern. This report, the second review of the public health impacts of global warming in California, builds on the literature review and conclusions reached in the 2006 report (Drechsler et al. 2006).

There have been a number of reviews on health-related issues related to climate change that take a fairly global perspective (e.g., IPCC, 2007; Ebi et al., 2008). The purpose of this paper is to summarize key findings on public health-related consequences of climate change that are relevant to California.

2.0 Approach

This paper is an update to the literature reviews in the 2006 scenarios analysis public health impacts report (Drechsler et al. 2006). It is based on a literature search performed with PubMed through December 2008. The review primarily focuses on studies performed in the United States, although papers from Europe or Australia that illustrate or explain key concepts are also included. Of the health endpoints that have been identified as being of concern with future climate change, heat-related mortality and morbidity have been most studied, generally with reference to specific heat waves. Other health endpoints identified in the 2006 report as of concern to California include air pollution, vector- and water-borne diseases, and wildfires, and the literature on these topics is also updated. The focus is on clinical health endpoints.

Key words used for the literature search included multiple permutations of: climate change, mortality, morbidity, hospitalization, heat, wild fires, vector-borne diseases, mosquito-borne diseases, tick-borne diseases, water-borne diseases, and food-borne diseases.

3.0 Results

This section summarizes the key conclusions on public health impacts that have been associated with ambient temperature.

3.1. Heat-Related Health Threats

3.1.1. Methodological Approaches and Challenges

There are several approaches to estimating the magnitude of temperature-related mortality and morbidity, along with a number of challenges. A key difficulty in estimating the number of cases of heat-related mortality stems from the lack of an "official" definition. Inconsistent definitions of heat-related mortality are applied between jurisdictions (Wolfe et al. 1999), and medical examiners frequently fail to consider heat as a cause of mortality when it is an uncommon diagnosis in their area. Heat-related mortality has been estimated in several ways, including counts based on death certificates, by comparison of observed deaths to expected deaths, and through application of time-series and case-crossover epidemiological models. Each of these methodologies addresses a different question, and it is difficult to compare studies that use different approaches. These methodologies are described below.

3.1.1.1 Directly Heat-related Deaths

The number of deaths due to heat is often estimated based on counts of death certificates where heat is listed either as the cause of, or as a directly contributing factor to death. These are often referred to as directly heat-related deaths because the direct cause of death is heat exposure.

3.1.1.2 Observed Compared to Expected Deaths

Estimates of excess deaths related to elevated ambient temperature are also made through comparison of the number of deaths during the time period of interest with that during a baseline period. This method defines heat-related deaths as this excess, regardless of the actual cause of death, and leads to estimates of heat-related mortality that are considerably larger than counts of directly heat-related deaths, as described above. However, in this method, most of the deaths are due to pre-existing chronic disease, and not necessarily due to heat-related causes (e.g., O'Neill et al. 2003).

A number of methodological choices influence the results of excess death analyses, making it often difficult to compare results between studies (Gosling et al., 2008). For example, calculation of expected (baseline) mortality is sensitive to the method used to estimate it. This in turn influences the calculated excess mortality, so that results from studies using different methodologies are not directly comparable. Additionally, the categories of deaths included in the analysis also influence the baseline mortality rate, which also makes it difficult to compare studies.

When long time periods are studied, failure to account for long-term trends, such as the changing age-structure of the population, changes and advances in health care, and other longer-term factors that influence vulnerability need to be accounted for to avoid bias in temporal comparisons. Other issues that influence comparison of studies include selection of the index of exposure, and consideration of factors that might modify or confound the relationship between ambient temperature and specific causes of death.

3.1.1.3 Time-Series and Case-Crossover Analyses of the Relationship between Temperature and Mortality in the United States

Retrospective analyses, typically covering several years, have investigated the relationship between high temperature and mortality in various regions of the United States. These analyses have generally used standard time-series epidemiological models, although some recent studies have used the case-crossover approach. The latter method is particularly useful for examining the association between a brief exposure and the acute onset of an adverse response. It is based on comparison of conditions on the day of death and those on referent days on which death did not occur, a method that allows cases to be their own controls (Basu et al. 2005).

These methods present challenges for analysis of long time-series, in that the methodologies are not able to account for long-term trends, such as the changing age-structure of the population, advances and changes in health care, and other longer-term changes that influence vulnerability and could bias temporal comparisons. As with the excess death approach, the index of exposure, and control for confounders, and potential effect modifiers can also influence the results of these types of studies.

These models allow investigation of the lag structure of the relationship between the time of heat stress and the advent of adverse effects. Lag refers to the time period between, for the present discussion, high temperature and an adverse health event. For example, a significant effect at lag 0 means that the temperature on the day of the adverse event is associated with the adverse outcome, while a significant association for lag 1 means that the effect is most associated with temperature on the day before the adverse event, etc.

3.1.1.4 Mortality Displacement

Several studies have investigated whether excess heat mortality represents mortality displacement (also called "harvesting"), which is defined as moving the time of death forward by a period of up to a few weeks in cases that would have occurred relatively soon in any event. The literature on whether or not exposure to elevated temperatures leads to mortality displacement reports somewhat inconsistent findings.

Braga et al. (2001) concluded from an analysis of 12 U.S. cities that excess heat-related mortality was primarily mortality displacement. Results suggesting some level of mortality displacement have also been reported in an 11 year study of 50 U.S. cities (Medina-Ramón & Schwartz,,2007), and in a study of the 1995 Chicago heat wave (Kaiser et al., 2007).

Rey et al. (2007) found evidence for mortality displacement in an analysis including all major heat waves that occurred in France between 1971 and 2003. Hajat et al. (2005, 2006) found significant short-term mortality displacement in London, but less in Delhi (Hajat et al. 2005); possibly because causes of death that are less heat-related, such as infectious diseases, still predominate in the latter city.

Several studies of the 2003 European heat wave also provide evidence that at least some deaths associated with heat waves represent mortality displacement, including in twelve French cities (Dear et al., 2005), and in 15 Mediterranean and north-continental European cities (Baccini et al. 2008). However, in contrast, analyses from Italy (Conti et al. 2007) and France (Fouillet et al. 2006; LeTertre et al. 2006) found no evidence for mortality displacement.

In summary, most studies of mortality displacement, both over several years and related to a specific heat wave, have found evidence for some level of mortality displacement, although the magnitude of the effect varies widely among studies. There are several possible explanations for these disparate findings, including the location of the study and the degree of heat adaptation in that population. It is also plausible that the duration, timing during the season and intensity of heat stress influence whether or not mortality displacement occurs. These factors have not been investigated to date. Another possible explanation of these disparate findings is provided by Gosling et al. (2007), who investigated mortality displacement in six international cities (two in the United States, three in Europe, and one in Australia) that have different climate patterns. The results suggest that a greater degree of mortality displacement may occur in cities that have heat mortality relationships with a higher threshold temperature and flatter slope, compared to cities with a lower threshold temperature and steeper slope. The results also suggest that mortality displacement was no longer evident 12 days after a heat wave.

3.1.2. Insights on the Heat-Mortality Relationship

3.1.2.1 Shape of the Temperature — Mortality Relationship

It is well known that temperature influences risk of mortality, and that in temperate regions the relationship between temperature and excess mortality is usually "U" or "J" shaped (Curriero et. al. 2002, 2003; Kunst et al. 1993; Davis et al. 2003a,b; Kysely 2004). The "trough" in the relationship represents a temperature threshold above and below which mortality increases. Although the general shape of this relationship has been reported for locations worldwide, threshold temperature, and the slopes of both the high and low temperature portions of the temperature-mortality relationship vary considerably among locations (Curriero et al. 2002; Keatinge et al. 2000b; Donaldson et al. 2003a; McGheehin and Mirabelli 2001; McMichael et al. 2008; Baccini et al. 2008). These studies demonstrate that high temperatures have a smaller effect on mortality in regions with typically hot summers compared to the same temperature in regions unaccustomed to high temperatures. This is demonstrated through the higher threshold temperature and the smaller slope of the high temperature arm of the temperature mortality relationship in hotter vs. cooler regions. In addition, these results lead to the conclusion that population responses to extreme temperature events, for example heat waves, and are most affected by the deviation from typical seasonal temperature. Moreover, this dependence on local climate conditions underscores the necessity of considering the temperature mortality relationship on a localized rather than large-scale basis.

Many questions remain as to the magnitude of temperature-related mortality. Several methodologies have been employed to estimate heat-mortality cases, although the available literature does not point to a standardized approach for investigating this topic. Each approach addresses different questions, and estimates derived from the different approaches vary. However, the consensus of the literature to date is that heat exposure does increase both mortality and morbidity. The next several sections summarize the insights gained from each analytical approach.

3.1.2.2 Directly Heat-Related Deaths

According to the Centers for Disease Control (CDC 2006), between 1999 and 2003 there were 3,442 deaths directly attributable to extreme heat exposure reported in the United States. This number includes 2,239 cases where excessive heat exposure was the direct cause of death,

although this is likely an underestimate due to under-reporting, and differences between localities in the criteria for ruling a death heat-related (Mirabelli and Richardson 2005; Basu et al. 2005). The remaining 1,203 cases had excessive heat exposure as a contributing cause of death. Of cases where age of the decedent was known, 7% were less than 15 years of age, 53% were between 15 and 64 years of age, and 40% were over 65 years of age. The states with the highest annual hyperthermia-related death rates during this time period were Arizona, Nevada, and Missouri.

When defined in this way, the majority of heat mortality cases are relatively young to middle-aged adults who were active in the heat prior to their demise, although a significant fraction of cases were older adults. The number of cases in children has been comparatively small, and often related to the child being trapped in a parked car. Among the 1,203 cases where hyperthermia was a contributing factor to death, cardiovascular disease was the underlying cause of death in 57%, while infection and psychiatric disorders accounted for 11%.

One of the largest subgroups with increased vulnerability to directly heat-related death is crop workers. The Centers for Disease Control (CDC 2008) reported that between 1992 and 2006, 432 crop workers died from exposure to environmental heat. Nearly all of these decedents were male, and 78% were between the ages of 20 and 54 years. Almost 60% of these deaths occurred in July, and most occurred in the afternoon. Twenty-one states have reported heat-related deaths among crop workers, but California, Florida and North Carolina accounted for 57% of all reported deaths. The highest rate of deaths in crop workers occurred in North Carolina. It is likely that this is an under-estimate of heat-related deaths in this subpopulation for the reasons noted above.

3.1.2.3 Excess Mortality

Excess mortality related to high ambient temperature has declined in recent years in both the United States and other regions. Davis et al. (2003b) found a decline in mortality rates related to high temperature in the 1980s and 1990s, compared to the 1960s and 1970s in 28 U.S. cities, although the reduction in temperature-related mortality was not uniform throughout the country. High temperature remained significantly related to mortality in the northeastern and north central parts of the country, while most southern cities no longer exhibited an increase in mortality rates on high temperature days by the 1990s. Further analysis of this data set (Davis et al. 2003a) showed that for the 28 cities as a group, annual excess heat-related death rates declined from about 41 per million people in the 1960s and 1970s, to 17 per million people in the 1980s, and to 11 per million people in the 1990s. The authors suggested that this decrease was due to the increasing use of air conditioning in the southern United States, improved health care, and heightened public awareness of the risks posed by elevated temperature.

Barnett (2007) investigated the influence of temperature on cardiovascular deaths in elderly Americans living in 107 U.S. cities by using daily cardiovascular mortality counts from the National Morbidity and Mortality Air Pollution Study (NMMAPS). The results showed that the average increase in cardiovascular deaths associated with a 5.5°C (10°F) increase in temperature was 4.7% in the summer of 1987, while by summer 2000 risk was -0.4%, suggesting either no effect of heat on mortality, or that heat provided a small benefit to survival.

Donaldson et al. (2003a) found that between 1971 and 1997 excess mortality related to temperature decreased in both North Carolina and South Finland, and remained unchanged in

Southeast England. This is in agreement with Carson et al. (2006), who reported that the percentage of all cause mortality attributable to heat was 0.4% in 1900–1910, and -0.9% in 1986–1996 in London, England.

3.1.2.4 Time-Series and Case-Crossover Studies

Basu et al (2005) compared the standard time-series approach with two case-crossover methods to estimate the influence of temperature on mortality in 20 U.S. cities (six in California) that were distributed throughout the country. The results were similar for the three different modeling strategies, and the odds ratio for heat-related mortality was greatest in the southwestern cities, which included Los Angeles, San Bernardino, Santa Ana, and San Diego. This study has several limitations that may explain why the results are not in agreement with those of others. The study included only one year of data, which was informative for addressing the main objective of the paper, which was to comparing several time-series and case-crossover analyses. In addition, data on some possibly relevant confounders or effect modifiers, such as air conditioner use, and socioeconomic status. Another factor could be that temperature exposure was based on a county-wide average because individual temperature exposure data were not available.

A four year retrospective analysis of the average relative risk of heat-related mortality for nine California counties (Basu et al., 2008) found a 2.3% (95% CI: 1.0–3.6) increase in mortality with a 5.5°C (10°F) increase in same-day mean apparent temperature (combination of temperature and humidity) using the case-crossover method, with a nearly identical relative risk using a time-series methodology. Relative risk of heat-related mortality was generally higher in the counties that had cooler climates. Further analysis of these data (Basu and Ostro 2008) examined factors that influenced vulnerability. The results showed increased risk for death for cardiovascular causes, especially ischemic heart disease. There was also increased risk for people over 65 years of age, infants less than one year of age, and for the African-American racial/ethnic group. Gender and educational attainment did not influence risk size. Medina-Ramón et al. (2006) has also identified African-American race as a risk factor for heat mortality.

A study using similar methods for nine U.S. cities (Zanobetti and Schwartz 2008) found a 1.8% (95% CI: 1.09–2.5) in mortality per 5.5°C (10°F) increase in apparent temperature using a case-crossover methodology, and a 2.7% (95% CI: 2.01–3.5) using a time-series methodology. The cities were in the eastern half of the United States, except for Phoenix, and had average summer apparent temperatures ranging from 20°C to 32°C (68°F to 90°F). Warmer cities had smaller effect estimates and/or wider confidence intervals than cooler cities. The results suggested that physiological and sociological acclimatization plays a role in reducing the heat effect in cities with a warmer climate compared to those with cooler climates.

Braga et al. (2001) carried out a six year time-series analysis for 12 U.S. cities to investigate the relationship between weather variables and acute mortality, along with the lag structure of the relationship. In the cities that had cooler weather patterns, both high and low temperatures were associated with increased deaths on the same or the preceding day. There was little effect of high or low temperature on deaths in the warmer cities, where the smaller effect of high temperature was attributed to the greater penetration of air conditioning in the warmer cities.

A case-cross-over study of the temperature-mortality relationship in 50 U.S. cities (Medina-Ramón and Schwartz, 2007) confirmed that heat increases risk of mortality, although the effect was quite heterogeneous among the cities, with the largest effects observed in cities with milder climates, less penetration of air conditioning, and higher population density. A case-only analysis for the same 50 U.S. cities (Medina-Ramón et al., 2006) found that the elderly, diabetics, African-Americans, less educated subjects, and those dying outside hospitals were more susceptible to death on extreme heat days.

These studies, drawing on different methodological approaches, support several conclusions: (1) there is a relationship between temperature and mortality even without extreme heat conditions; (2) the magnitude of risk relates to the typical local climate and is highly heterogeneous across the United States; and (3) some population sub-groups are more at risk than others. Below, additional insights are gathered from individual heat events.

3.1.3. Additional Insights from Historical Heat Waves

3.1.3.1 The 2003 European Heat Wave

The most extensively analyzed historical heat wave struck Europe for about 15 days in August 2003. This was the hottest period in Europe since 1500 (Poumadère et al. 2005). France, Italy, Spain, the United Kingdom, the Netherlands, and Switzerland all reported an increase in deaths (Table 1) during and shortly after this heat wave, although the death toll was highest in France.

Table 1. Deaths attributed to the 2003 European heat wave

Country	Region of Country	Excess Deaths	Change in Rate
France[a]	Whole country	14,802	+60%
Italy[b]	Whole country	7,659	+19%
England and Wales[c]	Whole country	2,139	+16%
The Netherlands[d]	Whole country	500	—
Switzerland[e]	Whole country	975	+7%
Portugal[f]	Mainland Portugal	1,316	+38%
Spain[g]	Provincial capitals	2,175	+17%

[a] Pirard et al. 2005; [b]Conti et al. 2007; [c] Johnson et al. 2005a,b; [d] Garssen et al. 2005; [e] Grize et al. 2005 (whole summer 2003); [f] Nogueira et al. 2005; [g]Simón et al. 2005

Multiple papers have examined the impact of the 2003 heat wave in France, which at the time had no public policy in place to address a heat emergency. The average Paris daytime high temperature during August is about 23°C (75°F) (weather.com), while during the 15 days from August 4 to 18, 2003, average daily high temperatures exceeded 35°C (95°F) (Canouï-Poitrine et al. 2006; LeTertre et al. 2006), and nighttime temperatures averaged about 3.5°C (6.3°F) higher than usual (Canouï-Poitrine et al. 2006). The unprecedented temperatures during this heat wave suggest what might result from an extreme heat wave with a lack of preparedness, including inadequate information for the public on effective protective practices and behaviors, absence of cooling centers, and minimal penetration of air conditioning into local building stock.

The increase in excess deaths was not uniform throughout the country. While almost 15,000 excess deaths are estimated to have occurred in France as a whole during this period, deaths were heterogeneously distributed across the country. The excess death rate for France as a

whole was about 60% higher than normal, although rates varied from a 36% increase in rural communities to a 140% increase in Paris (Canouï-Poitrine et al. 2006; LeTertre et al. 2006; Vandentorren et al. 2006). Even within the city of Paris, excess deaths were not evenly distributed, with the greatest number of excess deaths in the southern part of the city (Canouï-Poitrine et al. 2006). The increase in deaths lagged the start of the heat wave by one to three days (Vandentorren et al. 2006).

Although not mentioned in the literature reviewed here, several news media accounts point out that the heat wave occurred during the typical Parisian August vacation period. Many government officials were also away from Paris at the time of the heat wave. These factors led to slower government response to the emergency, and to disrupted social bonds and a reduced alternate safety net for the elderly who remained in Paris while family members were on vacation. (http://en.wikipedia.org/wiki/2003_European_heat_wave; http://www.cnn.com/2003/WORLD/europe/08/29/france.heatdeaths/).

The principal causes of death varied by age group, with directly heat-related deaths being much more common in younger, healthier individuals, while excess deaths were predominantly in the elderly. People 75 years of age and over accounted for 11,891 of the increased deaths in France, while there was little impact in children less than five years of age. Of the deaths in the over 75 years of age group, 2,852 were directly related to heat. The next largest fraction of deaths was from cardiovascular disease. Other frequent causes of death included undefined causes, respiratory diseases and diseases of the nervous system (Pirard et al. 2005; Fouillet et al. 2006; Poumadère et al. 2005). Among 2,565 deaths in people between 45 and 74 years of age, 434 were directly related to heat, while other deaths were due to undefined causes, cardiovascular diseases, cancer, mental illness, nervous system disorders, infectious diseases, and pulmonary diseases. There was a 19% increase in deaths of people under 45 years of age, which occurred principally in men, with the majority caused by undefined conditions, heatstroke, dehydration, and hyperthermia.

People less able to care for themselves were at increased risk of adverse outcomes, whether they lived at home or in retirement or nursing homes. A significant proportion of excess deaths occurred in Parisian nursing home patients, where the mortality rate was about four times higher than average during the heat wave (Holstein et al. 2005). These results suggested that level of dependency was a risk factor for death in these patients. Another study, Fouillet et al. (2006), found that excess mortality at home and in retirement institutions was greater than in hospitals. Belmin et al. (2007) also reported that level of dependency was a risk factor for heat-related death in community dwelling elderly people. Residents of institutions such as retirement and nursing homes are by definition less able to care for their own needs, and are often less able to recognize when they are at risk due to elevated ambient temperatures. The rarity of air conditioning in France, coupled with the frequently less robust health status of the elderly, and lack of planning for heat emergencies are all likely contributing factors to the increase in deaths in these sub-populations.

Several clinical factors on presentation at an emergency room were associated with increased risk of a poor outcome. Patients admitted to the emergency room who used antihypertensive medication or who had anuria, coma, or cardiovascular failure had poorer outcomes (Argaud et al. 2007). Follow-up with patients who survived heatstroke showed that they still had significant functional impairment one and two years later, and a substantial fraction had

become bedridden. In another study of patients evaluated in the emergency room of a Parisian hospital Davido et al. (2006) found that patients who had a core temperature ≥ 38°C (100.5°F) and/or with clinical signs of dehydration were at increased risk of death within one month. Other factors associated with a poor outcome were impaired ability to care for oneself, more serious clinical condition on presentation, alterations in blood chemistry indicative of disrupted physiological homeostasis, pre-existing ischemic cardiomyopathy, pneumonia, or previous treatment with psychotrophic medications.

Collectively, studies of the 2003 heat wave point to a number of factors that increase risk of heat-related mortality. These factors include age over 75 years, being female, and being single, particularly for men (Canouï-Poitrine et al. 2006; Poumadère et al. 2005; Pirard et al. 2005; Fouillet et al. 2006), poverty, isolation, living in an urban area (Poumadère et al. 2005), lack of mobility, pre-existing medical conditions, lack of insulation in the home, sleeping on the top floor of the building (directly under the roof), and an independent contribution from the heat island effect (Vandentorren et al. 2006). Poumadère et al. (2005) found that use of some medications commonly prescribed to treat cardiovascular disease and mental health conditions increased risk of adverse outcomes with heat exposure, a finding also reported in the U.S. (CDC, 2002; Kaiser et al., 2001). These medications, as a side effect, can reduce physiological thermoregulatory responses to heat through alteration of neurological control of the vasculature and sweat glands.

Similar findings have been reported for other European countries affected by the 2003 heat wave, including Spain (Simón et al. 2005), mainland Portugal (Nogueira et al.2005), Italy (Michelozzi et al. 2005a; 2005b), England and Wales (Johnson et al. 2005a,b; Kovats et al. 2006), the Netherlands (Garssen et al. 2005), and Switzerland (Grize et al. 2005).

3.1.3.2 U. S. Heat Waves

Heat wave analyses in the United States have focused primarily on heat waves that struck the Midwest during 1995 and 1999, particularly Chicago. A record-setting heat wave struck Chicago in July 1995, during which both daily maximum and minimum temperatures were unprecedented, and relative humidity was high (Semenza et al. 1996).

Medical examiner's records suggested that there were 437 directly heat-related deaths (Naughton et al. 2002), and at least 700 excess deaths (Semenza et al. 1996) attributable to the heat wave. Mortality peaked two days after the hottest day, 40°C (104°F), suggesting that there was a lag time between peak heat exposure and death (Kaiser et al. 2007). The most frequent causes of death were cardiovascular and heat-related. The number of deaths from respiratory disease did not change during the heat wave (Kaiser et al. 2007). Kaiser et al. (2007) reexamined the effects of the 1995 Chicago heat wave on all-cause and cause-specific mortality, using advanced time-series analysis methods that allowed for control of the meteorological and air pollution variables, and found results suggesting that about 26% of the observed deaths were due to mortality displacement.

Factors identified as being associated with increased risk of heat-related death included having chronic medical problems, being unable to care for oneself, not leaving home daily, living alone, living on the top floor of the building, having few social contacts, being bed-ridden, and lack of access to air conditioning, low educational attainment (i.e., less than high school), age above 75

years, African-American race, and chronic cardiovascular disease (O'Neill et al. 2003; Semenza et al. 1996; Medina-Ramón et al. 2006; Hajat et al. 2001; Kaiser et al. 2007).

Several factors reduced risk of heat-related mortality, including having regular social contacts, access to transportation, and having a working air conditioner (Semenza et al. 1996; Medina-Ramón et al. 2006; Hajat et al. 2001).

One response to the 1995 heat wave was development of an Extreme Weather Operations Plan for the Chicago area to disseminate information on how to avoid heat illness and death and to provide interventions focused primarily on the elderly to reduce risk of heat-related health conditions. Another heat wave struck the Midwest from July 29 through August 1, 1999, during which the new Extreme Weather Operations Plan was activated. About 80 excess heat-related deaths occurred during the three-day 1999 heat wave (Naughton et al. 2002), considerably fewer than occurred in 1995 (about 700 excess deaths). Fifty-three percent of the 1999 cases were less than 65 years of age.

Environmental conditions during the 1995 and 1999 Chicago heat waves were similar, and the large reduction in mortality in 1999 compared to 1995 has been attributed to the effectiveness of the Extreme Weather Operations Plan, and judged not likely due to meteorological differences between the two heat waves (Palecki et al. 2001). Both the 1995 and 1999 heat waves affected much of the Midwest, in addition to Chicago, a substantial reduction in morbidity and mortality was also reported for Milwaukee, which has been attributed to increased public health preparedness and response actions put into place after the 1995 heat wave (Weisskopf et al. 2002).

3.1.3.3 The California Heat Wave of 2006

A two-week heat wave occurred in California in July 2006. Historical analysis of major heat waves in California shows that they are typically characterized as either daytime or nighttime events, based on whether the daytime highs or nighttime lows are unusually elevated. The July 2006 heat wave had daytime temperatures that were high, although not record-breaking. Nighttime temperatures, however, during this heat wave were unprecedented (Gershunov 2007).

Examination of coroner's records found that 140 deaths were directly attributable to heat exposure during the heat wave, with cases concentrated in Imperial, Stanislaus, San Joaquin, Fresno, Kern, and Sacramento Counties (Kim and Trent 2007). This is likely an underestimate, for reasons discussed previously.

Evaluation of the characteristics of the decedents showed that about two-thirds were white, non-Hispanics, and about one-fourth were Hispanics. Two-thirds of cases were male. About 80% of cases were over 50 years of age, with only a few less than 20 years of age. However, among Hispanics almost half were less than 50 years of age. Of the 140 cases, ninety-six died indoors, and among these, 45% had no air conditioning. Thirty-five decedents had air conditioning units in their homes, but the unit was not functional in 16 cases, and not used in 18 cases. Almost half of cases lived alone, and among these, almost half had no known recent social contacts. These results and characteristics of decedents are similar to those described above for the Chicago heat wave.

Several causes of death predominated. Forty-seven percent had chronic cardiovascular disease, 23% psychiatric illnesses, 17% alcoholism, 7% chronic pulmonary disease, and 2% were confined to bed.

Knowlton et al. (2008) analyzed county-level hospitalizations and emergency department visits for all causes, and for grouped causes, and reported 16,166 emergency department visits and 1182 excess hospitalizations statewide during the heat wave. Children up to four years of age, and adults over 65 years of age were at greatest risk. Risk of hospitalization was highest in the Central Coast region of the state (including San Francisco), which has fairly cool summers. This underscores previous findings that risk of adverse effects with heat exposure is related more strongly to deviations from the usual temperature range than to the actual recorded temperature.

3.1.3.4 The Heat Island Effect

The heat island effect refers to the observation that ambient temperature is typically higher in urban areas than rural areas. This effect results from the tendency for greater heat absorption and retention in urban areas due to the high concentration of buildings, pavement, and roadways. There was evidence for the influence of the heat island effect on mortality in analyses of the 1995 Chicago heat wave (Naughton et al. 2002; Semenza et al. 1996), and the 1999 heat wave in the Midwestern U.S. (Palecki et al. 2001). Canouï-Poitrine et al. (2006) and Vandentorren et al.(2006) observed a greater effect of heat on mortality in urban compared to rural areas of France during the 2003 heat wave, as did Hajat et al. (2007) in England.

3.1.3.5 Additional Stresses from Air Pollution

It is well known that air pollution, especially ozone, is often highest on hot, sunny days, raising the question of the effect of co-exposure to high heat and air pollution on heat-related mortality. Most studies that have investigated the health effects of air pollutants such as ozone and particulate matter have reported that when the epidemiological models control for temperature there is little evidence that temperature confounds or modifies the effects of air pollution on health endpoints (see section 3.1.5.3). In contrast, when model control for air pollutants has been included in studies investigating heat-related effects, results have been inconsistent as to whether air pollutants modify or confound the relationship between temperature and health endpoints. While these results may appear contradictory, they demonstrate that while air pollution has an independent effect, the temperature effect is stronger.

Several older studies suggest that air pollution has a small confounding effect on the temperature mortality relationship (Rainham et al., 2003; Smoyer-Tomic and Rainham, 2001), although until fairly recently, little attention has been devoted to investigation of the potential for air pollution to confound or modify the relationship between temperature and mortality.

Fischer et al. (2004) and Stedman (2004) analyzed data from the 2003 European heat wave, and suggested that although heat-related mortality dominated the increased mortality observed, the proportion of deaths related to air pollution was greater than had been appreciated. These studies estimated deaths from concentration-response functions developed for analyses of air pollution effects, and are unlikely to adequately account for the influence of the heat wave on mortality. Levels of peak ozone and particulate matter 10 microns or smaller (PM_{10}) were not above California ambient air quality standards in the Netherlands (Fischer et al. 2004), and only slightly above California standards in the United Kingdom (Stedman 2004). The number of

deaths attributed to air pollution by these papers for the two-week period of the 2003 European heat wave is very high given the population size, the duration studied, and the pollutant concentrations.

Studies published since 2004 have produced inconsistent results as to whether air pollution confounds or modifies the relationship between ambient temperature and mortality. The joint effects of temperature and ozone on cardiovascular mortality were substantially heterogeneous across 95 U.S. cities in a study based on the National Morbidity, Mortality, and Air Pollution Study (NMMAPS) data set (Ren et al. 2008). In northern cities, ozone generally modified the association between temperature and cardiovascular mortality, but the effect was variable in southern communities. Effect modification or confounding was evident for Southern California communities, although when all 95 cities were combined in a Bayesian meta-analysis, ozone positively and significantly modified the temperature-cardiovascular mortality association. Baccini et al. (2008) and Filleul et al. (2006) also found significant heterogeneity in the influence of ozone on the temperature-mortality relationship in studies of 15 and nine European cities, respectively. Collectively, these papers suggest the influence of ozone on the temperature-cardiovascular mortality relationship tends to be smaller in populations regularly exposed to elevated ozone levels.

Several studies suggest that concomitant exposure to heat and ozone leads to higher risk of mortality than heat exposure alone. Dear et al. (2005) found that ozone significantly contributed to mortality during the August 2003 heat wave in France. A study from Sydney, Australia also found that ozone confounded the association between maximum temperature and mortality (Vaneckova et al. 2008a) during the six warm months of the year.

Several studies have found no evidence that ozone confounds or modifies the relationship between heat and mortality, including an analysis of the 1995 Chicago heat wave (Kaiser et al. 2007), and an analysis of nine French cities during the 2003 European heat wave (LeTertre et al. 2006). Keatinge and Donaldson (2006) investigated possible confounding of the relationship between mortality attributed to air pollution in the Greater London area using graphic and generalized additive modeling to include weather factors that have not typically been considered in these sorts of models. When temperatures were above 18°C (64°F), high levels of ozone were associated with sunshine, which increases heat stress. When confounding by sunshine was controlled for, there was no significant contribution of ozone to heat mortality.

Vaneckova et al. (2008b) used a synoptic (air mass) approach to investigate the relationship between weather and heat-related mortality over the warm seasons of 1993 to 2001 in Sydney, Australia. Days were classified into one of eleven different synoptic categories, and the correlation between these categories and heat-related mortality was estimated. Two synoptic categories were significantly associated with increased heat-related mortality. A relatively rare hot, dry category was most strongly associated with higher mortality rates, followed by a more frequent warm, humid category. Ozone was highest when the warm, humid category occurred, while PM10 occurred in high concentrations with the hot, dry category. Overall, however, the influence of the air pollutants on the relationship between synoptic pattern and mortality was unclear. O'Neill et al. (2003) also found inconsistent confounding by PM_{10}, in that the direction of the influence of PM_{10} (positive or negative) varied by location.

Two studies from Brisbane, Australia have investigated the effect of particulate matter exposure on the relationship between temperature and mortality. Ren et al. (2006) found that PM10 increased the effects of temperature on all non-external-cause mortality, and cardiovascular mortality at high concentrations. Ren and Tong (2006) found a statistically significant interaction between PM10 concentration and temperature on mortality, with a larger effect on warm days. However, the choice of the number of degrees of freedom used in adjusting for confounders, and the selection of arbitrary cut-offs for temperature affected the estimates of the size of the interaction, although the general conclusion held with all models evaluated.

A study from Sydney, Australia also found that PM10 confounded the association between maximum temperature and mortality (Vaneckova et al. 2008a) during the six warm months of the year.

Keatinge and Donaldson (2006) also evaluated the influence of PM10 using the same approach noted above for ozone. The results showed that at temperatures above 18°C (64°F), high levels of PM_{10} were associated with sunshine, and low wind, both of which increase heat stress. When confounding by sunshine and wind speed was accounted for, PM_{10} did not influence the relationship between temperature and mortality.

The inconsistent findings of these studies preclude a conclusion as to whether ozone and/or PM10 confound or modify the relationship between elevated temperature and mortality, and thus are prime areas for further research.

3.1.3.6 Heat Extremes and Mental Health

Few studies have investigated the impact of heat waves and high temperature on mental health. A series of studies performed in Adelaide, Australia found that mortality in people with mental and behavioral disorders increased during heat waves among people between 65 and 74 years of age, and also in persons with schizophrenia, schizotypal and delusional disorders (Hansen et al. 2008; Nitschke et al. 2007). While it may appear odd that people with mental health conditions are at increased risk of heat mortality, several factors contribute to increased vulnerability in this population. Many mental illnesses include some level of judgment impairment, the extent related to the nature and severity of the illness. In addition, some medications commonly used to treat mental health conditions affect functioning of the autonomic nervous system in ways that impair physiological temperature regulatory mechanisms, including redistribution of blood flow and sweating, so that the body is less able to thermoregulate.

3.1.4. Insights on the Heat — Morbidity Relationship

3.1.4.1 Background

In comparison to investigation of the relationship between high temperatures and mortality, few papers have been published on the impact of hot weather and heat waves on emergency room visits and hospitalizations, and other morbidities.

3.1.4.2 Preexisting Medical Conditions

Several studies suggest that the relationship between temperature and hospitalizations and emergency room visits for exacerbation of chronic disease is not parallel to that between temperature and mortality, and that it appears to have a shallower slope. Kovats et al. (2004a) examined the relationship between daily emergency hospital admissions and hot weather and

several heat waves between April 1994 and March 2000 in Greater London, England. There was no clear relationship between total emergency hospital admissions and high ambient temperatures, except for an increase in emergency admissions for respiratory and renal disease in children under 5 years of age, and for respiratory disease in people over 75 years of age. During a heat wave that occurred between July 29 and August 3, 1995 there was a 10.8% increase in daily mortality (95% CI: 2.8–19.3%), while there was only a small non-significant increase in hospitalizations (2.6%: 95% CI -2.2–7.6) during the same time period. These results suggest different relationships between morbidity and hot weather than between temperature and mortality, at least in the United Kingdom. The authors suggested that a possible explanation for these findings may be that many heat-related deaths may occur before the cases come to medical attention.

The influence of the intensity, duration, and timing of heat waves on hospital admissions over the summers of 2002 and 2003 in the Veneto Region of Italy in persons over 75 years of age was investigated by Mastrangelo et al. (2007). Admissions for heat-related disorders (including heat stroke, disorders of fluid and electrolyte balance and acute renal failure) and respiratory diseases increased with heat wave duration, but not intensity. The results suggest that at least four consecutive hot, humid days were required before there was a significant increase in hospital admissions. Interestingly, similar increases in hospitalizations occurred with the first heat wave of the season, in early June as the last, in August.

Several Australian studies of heat-related morbidity have investigated the relationship between indices of morbidity and heat. Although Nitschke et al. (2007) found no excess mortality during heat waves in metropolitan Adelaide, there were modest increases in calls for ambulance transport, and for hospitalization for mental health issues, causes related to renal function, and ischemic heart disease. A follow-up analysis (Hansen et al. 2008) found a positive association between ambient temperature and hospital admissions for mental and behavioral disorders above a threshold of 26.7°C. A third paper from this research group (Hansen et al. 2008) reported an increase in hospital admissions for renal disease and acute renal failure during heat wave compared to non-heat wave periods, although there was no relationship between heat and hospitalizations for dialysis, or for patients with co-morbid diabetes.

Several studies have investigated the relationship between cardiovascular morbidity and elevated ambient temperatures. Hospital admissions for heart disease in people over 65 years of age in 12 U.S. cities increased monotonically with same and previous day average temperature, with no evidence for an effect related to humidity (Schwartz et al., 2004). Kiu et al. (2004) compared coronary care unit admissions for atrial fibrillation on the 30 hottest and 30 coldest days of 2001 in South Australia. The results showed a smaller effect on hot days than cold. Michelozzi et al. (2008), in contrast, found no increase in cardiovascular hospitalizations, but instead, an increase for respiratory causes.

Morabito et al. (2005) used a biometeorological approach to investigate the relationship between weather and myocardial infarction in Florence, Italy for the period 1998 to 2002. Myocardial infarction was associated with number of discomfort days, and with days where apparent temperature was over the 90th percentile of the historical distribution for at least nine hours. Unusually high nighttime temperatures were also a contributing factor. The association between heat and hospitalization for myocardial infarction was strongest with a lag time of three days. Hot weather conditions increased the rate of hospital admissions in general, and

especially in young people. These results are in contrast to other studies suggesting that the greatest increase in hospitalizations is in the elderly.

One study has reported that in contrast to temperature-related mortality data, hospitalizations increased for respiratory, but not cardiovascular, causes as temperature increased. The study compared hospital admissions for cardiovascular, cerebrovascular, and respiratory causes during the warm season in people over 65 years of age in 12 European cities (Michelozzi et al. 2008). The strength of the association varied among the 12 cities, although it was evident in all the cities. The authors suggested that the variability in the strength of the association, which was evident among the hotter cities, may be influenced by between-country differences in delivery of health care, such as admission policies, and availability of hospital beds, along with socioeconomic status, and local customs of clinical management.

One paper has investigated the influence of elevated temperature on a neurological condition. Tataru et al. (2006) found no statistically significant relationship between mean ambient temperature and the number of hospital admissions or relapses in patients with multiple sclerosis in France during the 2003 European heat wave.

Larrieu et al. (2008) investigated morbidity in subsets of the participants in two established cohorts of elderly French people during the 2003 heat wave. The cohorts differed in the age of the subjects, and in the proportion of subjects that lived in urban compared to more rural areas. Subjects were questioned about their perceptions about their health during the heat wave, and for objective morbidity events, such as dizziness, fainting, falls, hospitalization or death, as well as behavioral changes they made to cope with the heat wave. During the heat wave, 8.8% of the subjects reported deterioration in their health, while 7.8% reported a clinically relevant outcome; however the subjects who reported deterioration in their health were not necessarily the same ones who experienced a clinically relevant event. Low educational level, being disabled, and being a woman increased risk of objective morbidity, while being able to ventilate the house and having a bathroom decreased risk of objective morbidity.

3.1.4.3 Additional Stresses from Air Pollution

It is well known that air pollution, especially ozone, is often highest on hot, sunny days, raising the question of the influence of co-exposure to high heat and air pollutants on heat-related morbidity. Only a few studies have considered how air pollution may influence heat-related morbidity.

Two studies from Brisbane, Australia have investigated the effect of particulate matter exposure on the relationship between temperature and adverse health impacts. Ren et al. (2006) found that PM10 increased the effect of temperature on respiratory and cardiovascular hospital admissions, although there was no clear evidence for an interactive effect on respiratory or cardiovascular emergency department visits. Ren and Tong (2006) found a statistically significant interaction between PM10 concentration and temperature on various indices of hospitalization, and emergency department visits, with a larger effect on warm days. However, the choice of the number of degrees of freedom used in adjusting for confounders, and the selection of arbitrary cut-offs for temperature affected the estimates of the size of the interaction, although the general conclusion held with all models employed.

An investigation of the influence of air pollution on the relationship between temperature and hospitalization for cardiovascular diseases among elderly people in Denver during July and August 1993 to 1997 was published by Koken et al. (2003). Once the relationship between temperature and hospitalization was modeled, the investigators introduced single air pollutants and evaluated whether air pollutants influenced the relationship between temperature and hospitalization. The results suggest that ozone is associated with an increase in risk of hospitalization for acute myocardial infarction, coronary atherosclerosis and pulmonary heart disease, in agreement with Linares and Díaz's (2007) results for Madrid, Spain. Koken et al. (2003) also found that particulate matter and nitrogen dioxide did not alter the relationship between temperature and any health outcomes. Sulfur dioxide increased the association between temperature and hospital stays for cardiac dysrhythmias, and carbon monoxide was significantly associated with congestive heart failure.

3.1.4.4 Conclusions on Heat Mortality and Morbidity

- The shape of the temperature-mortality relationship for extreme events differs from that for average seasonal temperature. Regions with hotter average temperatures have a higher threshold temperature for increased heat-related mortality, and the slope of the relationship between temperature and mortality is flatter, in contrast to areas with a cooler climate. Extreme heat events tend to have a smaller effect in areas with higher threshold temperatures.
- Heat mortality has a lag of 0-3 days.
- Different age groups are affected differently – causes of death vary by age. Directly heat-related deaths are more frequent in younger, healthier people than excess deaths, which occur predominantly among the elderly and people with chronic diseases.
- The wide variety of risk factors for heat-related mortality and morbidity identified to date includes personal characteristics and behavior choices, health status, socioeconomic, housing stock, and other factors. Since these factors touch on all areas of life, their variety presents challenges to development of public health interventions designed to promote population adaptation to future climate change.
- Hospitalizations related to heat predominately occur in the elderly and in people with chronic diseases.
- Incidence of heat-related morbidity does not parallel heat-related mortality.
- The influence of concomitant exposure to air pollution on heat-related mortality and morbidity remains unclear.

3.2. Cold-Related Health Threats

3.2.1. Background

Hypothermia, a preventable medical emergency, is a reduction in body temperature to less than 95°F (35.0°C). It is usually caused by prolonged exposure to cold temperatures without adequate clothing or other protection. The most common additional risk factors include advanced age, alcohol or drug use, altered mental status, and contact with substances that promote heat loss, such as water.

3.2.2. Methodological Approaches and Challenges

Similar approaches have been used to investigate cold-related mortality as were described for heat-related mortality. Fewer studies have investigated the contribution of low temperatures to mortality in the United States, than have investigated high temperatures.

3.2.3. Insights on the Cold-Mortality Relationship

3.2.3.1 Directly Cold-Related Mortality

Between 1979 and 2002, an average of 689 deaths per year was directly attributed to excess natural cold exposure in the United States. Using 2002 as an example (CDC 2005a), the majority of cold-related deaths occurred in males, and slightly over half in people over 65 years of age. The three key risk factors for directly cold-related mortality are advanced age, mental impairment (i.e., dementia), and substance abuse. The higher risk for older adults is believed to be related to reduced perception of the cold, and to a lower metabolic rate, which reduces body heat production. Persons with dementias and mental impairment due to substance abuse are particularly at risk due their lack of perception of the risks and dangers of cold exposure. Hypothermia-related death does not only occur under extreme conditions, and many cases are reported in areas with relatively mild cold weather. The U.S. annual death rate for hypothermia has declined since 1991 (CDC 2005a). While the subject of cold-related mortality may appear out of place in the context of global warming, it is essential to remember that research indicates that it will still play a role in total mortality even with increasing world temperatures.

3.2.3.2 Findings from Time-Series and Case-Crossover Analyses of the Relationship between Cold Temperatures and Mortality — U.S. Studies

Several studies have reported that cold-related mortality can occur not only during extremely cold days, but also at relatively mild temperatures. A study based on data from the National Morbidity and Mortality Air Pollution Study (Barnett 2007) that included data from 107 U.S. cities from 1987 to 2001 showed that although cardiovascular mortality was lower during warmer winters, the risk of cold-related mortality has persisted without significant change throughout the 14-year analysis period. In contrast, heat-related mortality declined during the same time period. In addition, the results showed that mortality risk not only increased on the coldest days of winter, but also was elevated on relatively cold days in spring and fall. The latter finding was confirmed by Cagle and Hubbard (2005), who reported a greater mortality rate for winter (0.64 per 100,000) than for summer (0.54 per 100,000) months in people over 55 years of age in King County, Washington, between 1980 and 2001, although the highest mortality rate was at temperatures below 5°C (41°F).

Braga et al. (2001) found that for 12 U.S. cities cold temperatures were most associated with deaths on the same day, although an increased number of deaths were observed over the following week or two, with no sign of a harvesting effect. The effect size varied among cities, and was larger in the warmer cities, in agreement with the results of Curriero et al. (2002, 2003).

Cold was most strongly associated with death from myocardial infarction and cardiac arrest, and was fairly homogeneous across 50 U.S. cities (Medina-Ramón and Schwartz, 2007) in a case-crossover study. The results showed a greater relative increase in risk of death for cardiovascular causes, and cardiac arrest on the coldest days. A case-only analysis for the same 50 U.S. cities (Medina-Ramón et al., 2006) also found that cardiovascular deaths showed the greatest relative increase on extremely cold days. They also found that susceptibility to death

related to temperature extremes varied for different primary causes of death. The results suggest that even though there is a wide range of winter temperatures in the U.S., the population seems well acclimatized to typical winter temperatures in their regions. The authors speculated that this may be due to the near universality of central heating in the U.S.

In contrast to the studies discussed above, Basu et al. (2005) found null or negative associations between temperature and mortality in the elderly during winter, spring, and autumn in the 20 largest U.S. cities. This difference in results may be related to several limitations of the study. The study included data for only one year, and information on several possibly important confounders and effect modifiers was not available.

3.2.3.3 Findings from Time-Series and Case-Crossover Analyses of the Relationship between Cold Temperatures and Mortality — European Studies

Perhaps the most comprehensive population-based study of cold-related mortality is the Eurowinter survey of cold-related mortality and protective measures employed in seven regions of Europe (North and South Finland, Athens, Baden-Württemberg, the Netherlands, London, and North Italy). These studies (The Eurowinter Group 1997; Keatinge et al. 2000a; Keatinge and Donaldson 2001; Keatinge 2002) found that cold-related mortality is more frequent in areas with milder winters than in areas with more severe winters, in agreement with U.S. studies (Barnett 2007; Curriero et al. 2002, 2003), and Analitis et al. (2008) in a study of 15 European cities. This somewhat counterintuitive finding is thought to be due to the greater understanding of the risk posed by cold, and preparedness to deal with it, among people who live in areas that frequently experience very cold temperatures. Table 3 shows the percentage increase in all-cause mortality per 1°C (1.8°F) when temperature falls below 18°C (64°F). Risk of cold-related mortality increased with age (The Eurowinter Group 1997; Keatinge et al. 2000a; Cordioli et al. 2000).

Table 2. Cold-related mortality in the Eurowinter study

City	Average Winter Temp. (°C)	% Increase in Mortality per 1°C Decrease in Temp.
N. Finland	-2.8	0.29
S. Finland	-1.0	0.27
Baden-Württemberg	5.1	0.60
Netherlands	6.2	0.59
London	7.6	1.37
North Italy	7.7	0.51
Athens	12.7	2.15
Palermo	15.4	1.54

Source: The Eurowinter Group 1997.

Several studies have investigated cold-related mortality in London (Keatinge and Donaldson 2001; Hajat et al. 2007; Donaldson and Keatinge 2003b). Keatinge and Donaldson (2001) found that atypical patterns of prolonged cold weather were associated with episodes of elevated air pollution, which could give false indications of mortality associated with sulfur dioxide, carbon

monoxide, or smoke when common epidemiological modeling techniques are used. The results underscore the importance of including all weather variables and lags that could impact the temperature-mortality relationship. In addition, a more extensive lag structure needs to be considered for cold-related mortality than heat-related mortality, because cardiovascular mortality related to cold occurs within about three days of minimum temperature, while cold-related respiratory mortality is associated with a lag of up to several weeks. The analyses showed no net increase in cold-related mortality related to current sulfur dioxide, carbon monoxide, or smoke concentrations in London. Monitoring data for particulate matter of any size range were too sparse to analyze.

There appears to be a 2–3 day lag time between cold exposure and cardiac events (Cagle and Hubbard 2005), while the lag is longer for respiratory deaths. Many excess winter deaths are due to influenza and other respiratory infections, possibly due to reduced building ventilation in cold weather and to the greater tendency for people to congregate in confined spaces, which increases the opportunity for cross-infection, and is consistent with the longer lag time in the association between cold exposure and respiratory mortality.

Hajat et al. (2007) found a relative risk for cold-related mortality in England and Wales of 1.06 per 1°C (1.8°F) decrease in temperature below the cold-related mortality threshold. Elderly people, particularly those in nursing or other care homes were at greatest risk of cold-related mortality, with risk increasing with advancing age. Respiratory and external causes of death accounted for the largest fractions of cold-related deaths. Among the population less than 65 years of age, cold-related mortality was primarily for causes related to chronic disease, that were not directly attributable to ambient temperature. The relationship between cold and mortality was not modified by deprivation, except for rural areas where the relative risk of cold-related mortality was slightly higher for more deprived people. An analysis of the influence of social class in working and retired age groups (Donaldson and Keatinge 2003b) found that men working in lower status, unskilled jobs, which generally involved greater physical activity, were at lower risk of cold-related mortality than men working in higher status positions, although the opposite was found for women. Risk of cold-related mortality in retired people tended to be higher in the lower social class compared to higher social class groups.

Pattenden et al. (2003) compared cold-related mortality in London, England, and Sofia, Bulgaria. Mean winter temperature in London was 8°C (12°F), and in Sofia, 3.9°C (7°F). For each 1°C (1.8°F) decrease in two-week average temperature below the local tenth percentile temperature, there was a 4.24% increase in mortality in London, and a 1.83% increase in Sofia, even though Sofia was colder than London.

Cold-related mortality is also influenced by home factors and personal behaviors. The Eurowinter study found that cold-related mortality varied inversely with the effectiveness of measures taken to avoid both indoor and outdoor cold, including such factors as home heating, the extent to which people regularly wore protective clothing and exercised while outdoors (The Eurowinter Group 1997). For example, people living in Finland, the coldest country studied, had better heating in their homes, dressed more protectively when outdoors, and were more physically active when outdoors than people living in the countries with warmer winters. These findings, supported by a study performed in Yakutsk, eastern Siberia (Donaldson et al. 1998) which found no increase in mortality related to cold even though the mean winter temperature for the period 1989 to 1995 was -26.6°C (-16°F), support the conclusion that many

cold-related deaths can be prevented with adequate home heating and insulation and by wearing proper clothing when outdoors.

Carson et al. (2006) reported that cold-related mortality in London has decreased steadily since 1900 for all causes, cardiovascular diseases, respiratory conditions, and non-cardiorespiratory causes. In the period 1900 to 1910, about 12.5% of all deaths in London were attributable to cold, while by 1986 to 1996 the attributable fraction was 5.42%. The authors posit that this reduction is related to improved housing, including home heating and improved insulation. The authors also suggest a role for increased car ownership, climate-controlled public transportation and shopping facilities, and improved clothing fabrics.

3.2.3.4 Conclusions

- Cold-related mortality is higher is areas with milder winters.
- Cold-related deaths are primarily due to cardiovascular or respiratory causes.
- Elderly people are at greatest risk.
- Cardiovascular deaths have a lag time of about three days, while respiratory deaths (primarily pneumonia and influenza) have a lag time of up to two weeks.

3.3. Air Pollution-Related Health Effects

3.3.1. Background

Exposure to various air pollutants, including ozone, particulate matter, nitrogen dioxide, sulfur dioxide, and carbon monoxide, has been associated with a variety of adverse health effects (CARB 2000). In this section, the focus is not on the confounding impacts of air pollution on heat or cold-related mortality and morbidity (as discussed above), but on the direct impacts of air pollution on human health. Health impacts of biogenic air pollutants (e.g., allergens) are not discussed, though they have been recognized as important climate-sensitive air pollutants that do and will increasingly affect human health (e.g., Kinney 2008; Ziska et al. 2008; Mohan et al. 2006). Because the available scientific literature suggests that ozone and PM are responsible for most of the health effects associated with criteria air pollutants, this review focuses on these two pollutants (Bernard et al. 2001; CARB 2000, 2002, 2005b).

Attainment of health-based ambient air quality standards is generally achieved through regulations that control emissions. Over the past 30 years, considerable progress has been made toward attainment of these health-based standards; however, many areas of California continue to be in non-attainment of the standards. State Implementation Plans (SIPs) have been developed to bring California into attainment with the federal ambient air quality standards. The current planning documents are available at:
http://www.arb.ca.gov/planning/sip/sip.htm.

Historically, climate change is not considered in the process of setting ambient air quality standards because the standards are based on health effects that are unrelated to ambient temperature. However, since many atmospheric chemical reactions contributing to the production of ozone and PM progress more rapidly at higher temperature, it is likely that all other factors remaining the same, the concentrations of these pollutants will increase, and that climate change could have an effect on the attainment process. Without additional emissions

control measures, climate change could slow progress toward attainment of the ambient air quality standards and increase control costs. The National Research Council (2004) recommended that "the air quality management system will need to ensure that pollution reduction strategies remain effective as the climate changes, because some forms of air pollution, such as ground-level ozone, might be exacerbated."

3.3.2. Ozone

Ground-level ozone is a secondary air pollutant that primarily forms in the atmosphere through a complex series of photochemical reactions between nitrogen oxides and reactive hydrocarbons. In urban areas, the principal source of these precursors is motor vehicles, along with the fuel supply system that supports them, although vegetation can contribute significant amounts of reactive hydrocarbons. Because ozone formation is highly dependent on solar radiation to drive the photochemical reactions, significant concentrations of ozone typically appear only during May through October, and during daylight hours, although peak concentrations occur later in the day in downwind locations because of transport (USEPA 2006).

Collectively, the literature on controlled exposure of human subjects to ozone indicates that one- to three-hour exposures to ozone concentrations as low as 0.12 parts per million (ppm) with moderate to heavy exercise can induce decrements in pulmonary function and increases in respiratory and/or ventilatory symptoms for some subjects. Increased airways reactivity and inflammation have been reported with one- to three-hour exposures to 0.40 and 0.18 ppm ozone, respectively. Concerns about the impacts of longer averaging times led to studies using a protocol that simulates a day of active outdoor work or play. These studies demonstrate that statistically significant group mean decrements in lung function, increases in symptoms of respiratory and/or ventilatory irritation, and increased airways reactivity and airways inflammation can be induced in the most sensitive fraction of the population with 6.6 to 8-hour exposures to ozone concentrations as low as 0.08 ppm (CARB 2005b).

Animal toxicological studies have shown that chronic ozone exposure can induce morphological changes similar to those characteristic of chronic lung disease (Last et al. 1994; Reiser et al. 1987; Harkema et al. 1993). Studies in primates suggest that intermittent ozone challenges with periods of clean air alternated with ozone exposure can lead to greater lung injury than daily exposures to similar concentrations (Tyler et al. 1988), while similar intermittent exposures in juvenile monkeys has demonstrated alterations in lung development (Evans et al., 2003; Schelegle et al. 2003). These data provide a biologically plausible basis for considering that repeated inflammation associated with exposure to ozone over a lifetime may lead to chronic lung injury.

Epidemiologic studies have shown positive associations between short-term ozone levels and several health effects including hospitalization (e.g., Burnett et al. 1997; Anderson et al. 1997), emergency room visits for asthma (e.g., Tolbert et al. 2000), restrictions in activity (Ostro and Rothschild 1989), respiratory symptoms, particularly in asthmatics (e.g., Gent et al. 2003), and school absenteeism (Gilliland et al. 2001). Many of these findings are observed or studied only in the summer season, when ozone levels are usually highest. A growing body of data now suggests that ozone concentration may represent an independent risk factor for premature death (Dominici 2003; Bell et al. 2004, 2005; Levy et al. 2005; Gryparis et al. 2004; Ito et al. 2005).

Long-term ozone exposure has been associated with respiratory inflammation (Kinney et al.1996), reduced lung function and respiratory symptoms (Kinney and Lippmann 2000), reduced growth of lung function in children (Galizia and Kinney 1999; Tager et al. 1998; Kunzli et al. 1997 , and asthma prevalence (Abbey et al. 1999; McConnell et al. 2002). Individuals at greatest risk of experiencing adverse health effects from ozone exposure are those who spend prolonged periods of time outdoors while participating in activities that increase the breathing rate, since ozone concentrations indoors are typically considerably lower than those outdoors. Highly exposed groups include children, outdoor workers, and recreational and professional athletes.

3.3.3. Particulate Matter

Particulate matter (PM) is emitted by many sources, and the size and chemical composition of particles can vary considerably among particles from different sources. Particles smaller than 10 microns in diameter (PM_{10}) can be inhaled deeply into the lung, and there is particular concern about particles 2.5 microns or less in diameter ($PM_{2.5}$) (USEPA 2004). Some particles are directly emitted from combustion processes, such as through combustion of gasoline or diesel fuel by motor vehicles, or generation of electrical power through combustion of oil, natural gas, or coal. Particulate matter is also emitted through a variety of industrial processes. Natural sources of PM include soil and dust. Some particles form through chemical reactions in the atmosphere. Examples of these particles include sulfates, nitrates, and organic aerosols (Seinfeld and Pandis 1998; Finlayson-Pitts and Pitts 1999).

The literature on health effects of PM was most recently reviewed by the U.S. Environmental Protection Agency during review of the federal ambient air quality standards for PM (U.S. EPA 2004), and by ARB as part of the Goods Movement Reduction Plan (CARB, 2006). The majority of literature on the public health impacts of particulate matter comes from epidemiologic studies. This literature fairly consistently reports statistically significant associations between changes in PM_{10} and $PM_{2.5}$ concentrations and a range of adverse health outcomes, both on a daily and a long-term basis. Associations between daily and long-term average changes in both PM_{10} and $PM_{2.5}$ and mortality appear to be independent of the effect of weather factors, seasonality, time, and day of week (Dockery et al. 1993; Pope et al. 1995, 2002, 2004; Krewski et al. 2000; Burnett and Goldberg 2003; Fairley 2003; Ito 2003; Laden et al. 2006; Jerrett et al. 2005; Ostro et al., 2008, 2007, 2006). Mortality occurs primarily in elderly populations, but has also been reported for infants (Romieu et al. 2004; Diaz et al. 2004; Kaiser et al. 2004; Ha et al. 2003; Bobek and Leon 1999; Loomis et al. 1999; Woodruff et al.1997). Studies over the past several years consistently report associations between PM_{10} and $PM_{2.5}$ and several different measures of hospitalization or urgent care for exacerbation of respiratory (i.e., chronic obstructive pulmonary disease, asthma) or cardiovascular diseases (i.e., congestive heart failure) (Atkinson et al. 2003; Zanobetti and Schwartz 2003; Sheppard 2003). These effects have been reported primarily among elderly individuals, but they have also been reported among all age groups, including children under age 18 (Ostro et al. 1999; Moolgavkar et al. 2000).

Data also suggest that long-term (i.e., months to years) PM exposures are associated with increased risk of mortality from cardiopulmonary causes (Dockery et al. 1993; Pope et al. 1995, 2002; Krewski et al.,2000; Laden et al. 2006; Jerrett et al. 2005). Associations with PM_{10} and $PM_{2.5}$ exposure have also been reported for chronic respiratory symptoms or disease, possibly decreased lung function in general (e.g., Ferris et al. 1973, Hodgkin et al. 1984; Mullahy and

Portney 1990), and with prenatal and lifetime exposure in asthmatic children (Mortimer et al. 2008).

3.3.4. Conclusions

- Both ozone and particulate matter have been associated with premature mortality and morbidity, including hospitalizations, emergency room visits, respiratory symptoms, school absences and work loss days.
- The most at-risk populations include the elderly, children, and people who are active outdoors, including outdoor workers and athletes.
- Federal and State ambient air quality standards are health-based, and are promulgated to protect public health. Because climate change will make attainment of ambient air quality standards more difficult, both federal and State actions to achieve attainment will need to continue. Consequently, the extent of future air pollution-related adverse health effects will be related to the extent to which attainment efforts succeed or fall short.

3.4. Wildfires and Public Health

3.4.1. Background

Wildfires can be a significant contributor to air pollution in both urban and rural areas, and they have the potential to significantly affect public health primarily through their smoke. Fires also affect the economy and public safety. Various climate change scenarios project that through the twenty-first century there will likely be an increase in the frequency, size, and intensity of wildfires (Westerling and Bryant 2006; Westerling et al., 2009). However, quantitative estimation of the public health impacts of future wildfire events is extremely difficult. The public health impacts of any fire are unique to that fire, and are influenced not only by the magnitude, intensity, and duration of the fire, but also the proximity of the smoke plume to a population.

Smoke from burning vegetation includes a large quantity of $PM_{2.5}$ and PM_{10} (Lee et al. 2008; Viswanathan et al. 2006). Schöllnberger et al. (2002) have shown that the particulate matter in wildfire smoke deposits throughout the respiratory tract, with deposition fractions of 54%–58% for particles less than 0.02 m in diameter, and 76%–78% for particles less than 10 m in diameter. Adult men and women had total deposition efficiencies at the lower end, and newborn infants had deposition efficiencies at the upper end of these ranges. Wildfire smoke impacts on public health can reasonably be inferred from the extensive literature on the health impacts of $PM_{2.5}$ and PM_{10}, which was discussed above in the section on air pollution-related health effects.

3.4.2. Methodological Challenges

Because wildfires are relatively rare, and most frequently occur in sparsely inhabited areas, there are few studies evaluating the public health impacts of wildfires. Assessment of the public health impacts of wildfires is also complicated by the fact that there are usually few, if any, air quality data available for fire-impacted regions. Since this precludes development of exposure assessments and concentration-response functions, analyses of public health impacts of wildfires are generally limited to retrospective assessments of the change in incidence of health endpoints during the fire compared to that observed during a baseline time period.

The largest group of studies has examined changes in emergency room visits or self-reported symptoms. However, because the decision to visit an emergency room is influenced by individual perceptions of risk and a decision to seek medical care is not necessarily based on objective assessment of medical need, these results are somewhat subjective. In addition, psychosomatic stress associated with wildfires and resulting air quality emergencies also can influence the decision to visit an emergency room (Mott et al. 2005; Lipsett et al. 1994). There are few studies of more objective and severe health endpoints, such as hospitalization and mortality, primarily because population densities in exposed communities are rarely large enough to yield sufficient sample sizes for assessment of epidemiologic relationships between poor air quality due to fires and health endpoints (Vedal and Dutton 2006), and because adequate air quality data are generally not available (Mott et al. 2005).

3.4.2.1 Wildfires and Mortality

There have been few investigations into the impact of wildfire smoke on mortality (non-burn-related). While not directly similar to the sort of fires that occur in California, two reports on a series of fires in Southeast Asia in 1997 suggest a combination of factors that created a particularly severe and long lasting smoke exposure scenario. The few air quality data available suggest that PM_{10} levels varied widely during the fire period. Many days had typical PM_{10} concentrations, but there were days on which PM_{10} concentrations reached alarming levels. Between August and early November 1997, Singapore air quality reached the unhealthy range, based on the U.S. Environmental Protection Agency Pollutant Standards Index (PSI; values over 100 are considered unhealthy) on 12 days, with the highest PSI recorded being 138. Monthly average PM_{10} values, typically between 30 and 50 micrograms per cubic meter (g/m^3), increased significantly to 60 to 100 g/m^3 during September and October 1997 (Emmanuel 2000), although there was no significant change in mortality with an increase in PM_{10} from 50 to 150 g/m^3. Sastry (2002) found a 19% increase in mortality ($P<0.05$) in Kuala Lumpur, Malaysia on the day following days with 24-hour average PM_{10} measurements above 210 g/m^3 in people 65 to 74 years of age, but not in other age groups at any PM_{10} concentration. The ninetieth percentile PM_{10} value was 99.4 g/m^3. The study estimated a relative risk of mortality with a 100 g/m^3 increase in PM_{10} of 1.07. The results also suggested that displacement of deaths from the exposure to the smoke haze was short lived, although for those aged 65 to 74 there was an upward shift in death rate that lasted for a few weeks after the fires ended. Overall, the study found that mortality burden, in terms of days of life lost, was small.

Vedal and Dutton (2006) evaluated the relationship between smoke, measured as $PM_{2.5}$, and mortality consequent to a wildfire near Denver, Colorado (Denver metropolitan area population was about two million at the time of the study). On two days in June 2002 peak one-hour PM_{10} and $PM_{2.5}$ concentrations reached 372 g/m^3 and 200 g/m^3, respectively on June 9th, and 316 g/m^3 and 200 g/m^3, respectively, on June 18th, but there was no evidence for an increase in daily mortality attributable to these fire-related increases in PM_{10} or $PM_{2.5}$ level. Although not a wildfire, Lipsett et al.'s (1994) found no observable increase in respiratory mortality related to a supermarket warehouse fire that occurred in Richmond, California, in July 1988. Although not the usual sort of wildfire, these results are relevant, in that wildfires in California often spread into urban areas, adding combustion emissions to the smoke plume from building materials and household and consumer goods to those of vegetation. Analysis of coroner's records following an urban wildfire that burned parts of Alameda County, California, in late October

1991 (Shusterman et al.1993), found 25 fire-related deaths, all principally due to extensive burns, although many patients also had smoke inhalation injury.

3.4.2.2 Wildfires and Morbidity

Several studies of the impacts of a series of fires in 1997 in Southeast Asia have reported on morbidity endpoints, including increased respiratory symptoms, upper respiratory tract illnesses, and hospitalizations. As noted above, few air quality monitoring data were recorded during the fires PM_{10} levels varied widely during the fire period, and reached very high levels on some days. Emmanuel (2000) found that an increase in PM_{10} from 50 to 150 g/m^3 was associated with a 12% increase in upper respiratory tract illness, a 19% increase in asthma exacerbation, and a 26% increase in rhinitis. However, these increases were not coupled with a significant increase in hospital admissions. In contrast, Mott et al. (2005) found an 8% increase in hospitalization rate for all causes compared to baseline, with the greatest increase for respiratory hospitalizations between August 1 and October 31, 1997, in the Kuching region of the Malaysian state of Sarawak. The number of persons over 65 years of age readmitted to the hospital during the fire period was significantly higher than expected, and re-hospitalization rates returned to the pre-fire level once the fires ended.

Several studies have investigated the public health impacts of wildfires in the western United States. Vedal (2003) reported an increase in the mean number of daily emergency room visits for respiratory causes on two days in June of 2002 when peak one-hour $PM_{2.5}$ concentrations reached 352 and 390 g/m^3, consequent to a wildfire near Denver, Colorado. Morris et al. (2003) found that those who were not evacuated from the area affected by smoke from the June 2002 Rodeo-Chedeski fire in Arizona reported significantly more respiratory symptoms than people who were evacuated. The most commonly reported symptoms were eye irritation, itchy sore throat, and cough. Prevalence of self-reported asthma exacerbation increased 86% among people living in the non-evacuated area, compared to 39% among evacuees. Sutherland (2005) followed a small panel of chronic obstructive pulmonary disease (COPD) patients living in Denver during the 2002 fire. There was a small increase in symptom scores on the days with the highest $PM_{2.5}$ concentrations, followed by return to baseline on the following, lower $PM_{2.5}$ day. Unfortunately, there were no objective measures of lung function, and since lung function changes and symptoms are not well correlated in COPD, the clinical and functional significance of the small increase in symptoms is unknown.

Several studies have investigated morbidity related to fires in California. Duclos et al. (1990) assessed the impact of a series of fires in late summer of 1987 that burned over 600,000 acres of California forests. There was a 40% increase above expected in the number of emergency room visits for asthma (120 vs. 86) and a 30% increase in visits for chronic obstructive pulmonary disease (74 vs. 57) during the period with the highest smoke levels. Analysis of emergency room visits to nine local hospitals during the two days of an urban wildfire that burned parts of Alameda County, California, in late October 1991 (Shusterman et al.1993) showed that smoke inhalation was the principal cause of about one-half of the emergency room visits during the fire. Lipsett et al. (1994) also reported an increase in emergency room visits for asthma and other lower respiratory conditions and for respiratory-related hospitalizations during a supermarket warehouse fire that occurred in Richmond, California, in July 1988.

In October of 2003 a series of wild fires burned a large portion of Southern California, including several Children's Health Study sites, providing a serendipitous opportunity to investigate the impact of wildfire smoke on the respiratory health of children (Künzli et al. 2006). Exposure to wildfire smoke was associated with increased eye and respiratory symptoms, medication use by asthmatics, and unscheduled physician visits. Symptoms were more severe with both higher levels of smoke and more smoky days. Surprisingly, the associations were strongest for children without asthma. Children with asthma were more likely to take preventive action, likely explaining the lower level of adverse effects in the asthmatic compared to the non-asthmatic children. This is the first study to report benefits of preventive actions, including remaining indoors, reducing physical activity, using air conditioning, and wearing masks when outdoors.

In 1999 a large wildfire burned from August 23 to November 3 near the Hoopa Valley National Indian Reservation in Northern California. On 15 days, the PM_{10} concentration exceeded the U.S. Environmental Protection Agency (U.S. EPA) 24-hour standard of 150 g/m^3, and on October 21 and 22, PM_{10} levels exceeded the U.S. EPA hazardous level of 500 g/m^3. A survey of 26% of tribal households showed a 52% increase in medical visits for respiratory problems during the fire, compared to the same period of 1998. More than 60% of those surveyed reported an increase in respiratory symptoms during the smoke episode, and 20% continued to report increased respiratory symptoms two weeks after the smoke cleared (Mott et al. 2002).

3.4.3. Conclusions:

- The majority of studies of the health effects of wildfires focus on respiratory symptoms and emergency room visits.
- Since fire smoke produces particulate matter in the PM2.5 size range, health effects would be expected to be comparable to similar concentrations of PM2.5, with similar exposure durations.
- Exposures to fire smoke for up to a few days induce respiratory and ocular symptoms, but the limited data available suggest that few serious effects would be expected.

3.5. Infectious Diseases

3.5.1. Water-Borne Diseases

Available data show that meteorological factors, such as ambient temperature and precipitation, can influence the occurrence of outbreaks of water-borne diseases. Water quality and safety concerns apply not only to the drinking water supply, but also to irrigation and recreational waters, since all can be sources of human illness due to water-borne pathogens (Lipp et al. 2002; Hunter 2003; Rose et al. 2000). Exposure to these pathogens can occur through the drinking water supply, with failure at various points throughout the delivery system, and through contact with contaminated ground or surface water (Liang et al. 2006). Water-borne diseases can be acquired through ingestion, or by physical contact with contaminated water.

Historical data link many outbreaks of water-borne disease to water contamination subsequent to runoff from heavy rainfall, flooding, and/or sewage overflow (Curriero et al. 2001, 2003; Charron et al. 2004; Colwell and Patz 1998; Louis et al. 2003; Auld et al. 2004; Lee et al. 2002; Rose et al. 2000). For example, there was a doubling in the rate of gastrointestinal illness following floods related to Hurricane Floyd among residents in the eastern part of North

Carolina (Setzer and Domino, 2004). Infections were linked to pathogens originating from the high concentration of hog farms in the area, and illustrate how widespread flooding can spread disease-causing pathogens to downstream areas. Another illustration of the extent to which flooding can lead to serious disease outbreak occurred in Walkerton, Ontario in May 2000 (Auld et al. 2004; Hrudey et al. 2003). Rainfall, accompanied by record high temperatures, exceeded the 60 to 100 year event marks in the region surrounding this small town. Almost half of the local population became seriously ill, and seven died from ingestion of E. Coli and Campylobacter contaminated drinking water. Wade et al. (2004) found that contact with floodwater from the Mississippi River during a severe flooding event in 2001 was related to gastrointestinal symptoms and illness, although there was no relationship between tap water consumption and illness.

Flooding can also contaminate ground water, in addition to surface waters, leading to outbreaks of water-borne disease (Liang et al 2006). For example, groundwater can become tainted through contamination of the surface waters that feed the aquifers, as well as through leakage from septic systems, sewage overflows, and dumps. Contamination can also occur after water treatment if treatment is insufficient for the pathogen load, or if the water delivery system is breeched (Rose et al. 2001). Irrigation water can become contaminated by similar sources and from flooding and storm runoff, particularly if the runoff contains livestock excrement (King and Monis 2006).

The most common water-borne illness is gastroenteritis, followed by dermatitis. These diseases are under-reported, and the majority of cases are self-limited and do not come to medical attention. However, a fraction of cases require medical attention or hospitalization, and there have been some deaths related to water-borne pathogens reported in the United States (Dzuiban et al. 2006; Lee et al. 2002; Liang et al. 2006).

The most commonly isolated causal agent in drinking water has been the bacterium Cryptosporidium, although a significant number of cases have also been associated with various Vibrio species, and giardia (Dzuiban et al. 2006; Furness et al. 2000). Liang et al. (2006) have reported a significant number of cases due to Legionella spp. associated with water that was contaminated in the drinking water distribution system.

Outbreaks of gastroenteritis associated with contaminated recreational waters have been commonly associated with Cryptosporidium parvum in treated recreational water such as swimming pools, and E.Coli in freshwater venues (Lee et al. 2002). Several bacteria, viruses and parasites, including Campylobacter (Dzuiban et al. 2006), Cryptosporidium (Lee et al. 2002), Vibrio species (e.g., Morris, 2003; Randa et al. 2004; Dzuiban et al. 2006; Furness et al. 2000), leptospirosis (Meites et al. 2004), and the amoeboflagellate Naegleria fowleri (Marciano-Cabral et al. 2003) have been linked to outbreaks of waterborne diseases associated with untreated recreational water.

Leptospirosis has recently been identifies as a re-emerging zoonosis, after being eliminated from the list of reportable diseases in 1995 (Katz et al. 2002). Hawaii has consistently had the highest incidence rate, with cases predominantly in males. Cases related to occupational exposure have declined in recent years, while cases related to recreational water have increased.

Water-borne illnesses are more common during the warmer months of the year (Dzuiban et al. 2006; Furness et al. 2000; Janda et al. 1988), suggesting a link to ambient temperature. Several studies have evaluated the influence of water characteristics on Vibrio species. High concentrations of these species and outbreaks of water-borne disease related to Vibrio species are more frequently associated with warmer water (Janda et al. 1988; Pfeffer et al. 2003; Randa et al 2004; Thompson et al. 2004) and lower water salinity (Lipp et al. 2002; Louis et al. 2003; Randa et al. 2004), turbidity (Pfeffer et al. 2003), and level of dissolved oxygen (Pfeffer et al. 2003). Campylobacter infections from non-food sources have also been found to correlate with temperature (Louis et al. 2005). Environmental factors have also been associated with survival of Cryptosporidium oocytes (King and Monis 2006).

At-risk populations identified include the immunocompromised exposed to contaminated surface water (King and Monis, 2006), children and adults between 31 and 40 exposed to recreational water contaminated with giardia (Furness et al. 2000), children exposed to flood waters (Wade et al. 2004). Although not specifically addressed by the literature cited above, children and the elderly are more likely to require hospitalization for gastroenteritis, regardless of the cause, than other sub-populations (Charles et al. 2006; Gangarosa et al. 1992; Louis et al. 2005).

3.5.2. Food-Borne Diseases

Food-borne illnesses are a significant public health concern worldwide. These illnesses can be caused by a variety of microorganisms, including the pathogens discussed above under water-borne diseases. Other pathogens related to food-borne diseases include Listeria, Salmonella, E. Coli, Shigella, and Yersinia (Vugia et al. 2006). These pathogens typically cause gastroenteritis (Lynch et al. 2006). Many cases of food-borne disease are mild and self-limited, but food-borne illnesses cause a significant number of hospitalizations and deaths annually (Mead et al. 1999).

Several studies have found that ambient temperature influences the number of food-borne disease reports. D'Souza et al. (2004) has reported that seasonal patterns in salmonellosis reports can be explained by changes in temperature. The results also suggest that elevated temperature during earlier phases of the food production pathway may have a greater influence on whether or not food becomes infected with salmonella than at the food preparation phase. A similar linear association between temperature above 6°C and reported cases of salmonellosis has been reported for a number of European countries, with the strongest association for adults 15 to 64 years of age (Kovats et al. 2004), and for temperature one week before the onset of illness. Kovats et al. (2005) have also investigated the influence of ambient temperature on campylobacter infection. The results of this analysis indicated a distinct seasonality in campylobacter transmission, along with geographical variation in the time of seasonal peak activity that suggests that climate may be a contributing factor in disease transmission. Zhuang et al. (1995) have reported that the population of Salmonella Montevideo on tomatoes, both whole and chopped, increased significantly as storage temperature increased.

Seafood caught in waters infected with various species of Vibrio is a fairly common source of food-borne illness, in fact, seafood ranked third on the list of causes of food-borne disease in the U.S. between 1983 and 1992 (Lipp and Rose 1997). Raw oysters are one of the more common sources of Vibrio infection, and water temperature has been related to oyster contamination and subsequent disease (Morris 2003. Since 1997 mean Alaskan sea water temperature has

increased, leading to a significant outbreak of Vibrio poisoning that was traced to Alaskan oysters that had been harvested when mean daily water temperatures exceeded 15°C (McLaughlin et al. 2005). Several viruses associated with fecal contamination, and toxins related to toxic algal blooms have also been responsible for outbreaks of seafood-related food-borne illness (Lipp and Rose 1997).

3.5.3. Other Infectious Diseases

Several other infectious diseases that are spread by oral-fecal or respiratory routes have patterns of seasonality.

Diarrheal illness is common in the United States, with estimates of approximately 200 million episodes each year (Herikstad et al. 2002). Gastroenteritis associated with rotaviruses is primarily a winter disease in temperate climates (Charles et al. 2006; Cook et al. 1990). Fleury et al. (2006) have reported a strong non-linear association between ambient temperature and occurrence of Salmonella, E. Coli and Campylobacter infections in Alberta, Canada, and Campylobacter in Newfoundland-Labrador. Enteroviruses, including the family of echo viruses and coxsackie viruses, also have a seasonal pattern of infection; the greatest number of confirmed cases occurring between June and October (Khetsuriani et al. 2006). There appears to be a two to fourteen day lag between peak ambient temperature and peak incidence of Campylobacter and Salmonella infection, while Cryptosporidium, Shigella and Giargia infections peaked about 40 days relative to peak temperature (Naumova et al. 2006). Risk of hospitalization and death related to diarrheal disease is widely recognized for young children. However, the risk to the elderly, among whom the case-fatality ratio is higher than for children, has not been widely appreciated (Gangarosa et al. 1992).

Several respiratory diseases also have distinct seasonality. Several studies have investigated the influence of season on influenza. Although the influenza season is typically during the colder part of the year, cold weather alone does not predict influenza deaths (Dushoff et al. 2005).

Several papers have investigated the influence of El Niño on respiratory diseases. Both the circulating subtype and magnitude of the El Niño Southern Oscillation (ENSO) are associated with the impact of influenza epidemics (Viboud et al. 2004). Choi et al. (2006), in an analysis focused in California, found that mean influenza-related mortality was lower during non-ENSO periods, compared to El Niño periods. An analysis of the influence of El Niño on hospitalization for viral pneumonia in females in six California counties (Ebi et al. 2001) found that temperature had an inconsistent relationship with hospitalization among the various counties. There was a significant increase in hospitalizations in San Francisco and Los Angeles with increasing temperature, with the association independent of season. The results also suggested that viral pneumonia could continue to be a public health issue as climate warms. El Niño was only associated with hospitalizations in Sacramento, with a significant decrease for girls, and an increase for women.

Two papers have investigated the influence of climate conditions on incidence of valley fever, which is endemic to arid regions of the Western Hemisphere, and is caused by a soil-dwelling fungus. Antecedent temperature and precipitation in are important predictors of incidence (Kolivras and Comrie 2003). The results suggested that temperature and precipitation in the winter season lagged by a year or more were most predictive of incidence. In contrast,

Talamantes et al. (2007) found that fluctuations in incidence of valley fever were related to biological and/or anthropogenic factors, and only to a small extent to weather anomalies.

3.5.4. Vector-Borne Diseases

Vector-borne diseases are caused by a variety of viruses, bacteria, and protozoa that spend part of their life cycle in a host species (mosquitoes, fleas, and ticks) and are spread to humans and animals during insect feeding. Although twelve mosquito-borne diseases are known to occur in California, only Western Equine Encephalitis (WEE), West Nile Virus (WNV), and St. Louis Encephalitis Virus (SLV) are significant causes of human disease. Currently, West Nile Virus is the most important in California. These diseases are maintained through a cycle that depends of wild birds and mosquitoes, with humans as an incidental host.

The primary mosquito vector in California appears to be *Culex tarsalis*, although other sub-species of mosquitoes can also carry these diseases (California Department of Health Services 2004). The *Culex* family of mosquitoes is unique in that it is urban dwelling and prefers to breed in foul water such as that found in storm drains and catch basins (Epstein 2001). Consequently, drought tends to increase the richness of rotting organic material in storm drains, improving the mosquito habitat, while heavy rains flush the drains, and reduce the quality of the habitat leading to reduction in vector survival. Reisen et al. (2006) has reported that warm temperatures are necessary for virus development, that the zero virus development level is approximately 14.3°C, and that during the epidemic summers of 2002 to 2004, WNV dispersal and epicenters were closely linked to above average summer temperatures. An investigation of the influence of weather parameters on mosquito abundance and WEE transmission in Kern County California showed that the water content of snow in the Sierra Nevada during winter was correlated with spring river runoff, mosquito abundance and WEE activity in the San Joaquin Valley (Wegbreit and Reisen 2000). The strongest predictor of host-seeking females collected per trap night per month during summer was river runoff one month earlier.

DeGaetano (2005) found that between June and August, climatological conditions accounted for between 40% and 50% of the variation in the number of trapped *Culex* mosquitoes in two metropolitan New Jersey counties. In central Illinois, the *Culex* pipiens mosquito is the principal carrier of West Nile virus, although *Culex* restuans Theobald is also endemic (Kunkel et al. 2006). The time at which the two species of *Culex* are in equal abundance (crossover) marks the beginning of an increase in infections. This crossover time varies from year to year, and is influenced by temperature, in that warmer temperatures are correlated with earlier crossover dates.

The most common tick-borne disease in the United States is Lyme disease, caused by the bacteria *Borrelia burgdorferi*, and transmitted in the western United States by the *Ixodes pacificus* tick. Seventy-six cases of Lyme disease were reported in California in 2007, covering 23 counties (CDPH 2007). The primary hosts for this disease are deer and mice, and risk of infection increases in proportion to the populations of each (Gubler et al. 2001). Lindgren et al. (2000) reported a northern shift in the distribution of ticks related to fewer very cold days during the winter season in Sweden. Although the ticks studied are not of the same species as that seen in California, the results of the analysis support the notion that a vector's geographical range can alter with changing ambient temperature and humidity, as well the availability of suitable host

species (i.e., deer and mice), which can also be affected by climate and weather conditions (Gubler et al. 2001).

A series of studies by Ogden et al. (2004; 2005; 2006a; 2006b) investigated the influence of temperature and other weather-related factors on development of *Ixodes scapularis* ticks, and the potential for them to spread into currently non-endemic regions of Canada. Although this species of tick is not found in California, where Lyme disease is spread by the *Ixodes pacificus* tick, the findings of this series of studies are illustrative of the complex interaction between the life cycle of ticks and environmental factors. Ogden et al. (2004) found that while temperature affected several developmental phases of the tick life cycle, other phases were more dependent on availability of a suitable host for obtaining a blood meal or on length of daylight. It was also clear that at temperatures above 30° C few female ticks produced viable eggs. Ogden et al. (2005) found that, based solely on temperature, it was possible to apply a dynamic population model and predict areas of Canada that were conducive to establishment of tick populations. It is important to note that not all areas that met the temperature criteria for supporting tick populations in fact had tick populations, and that factors other than temperature affect tick viability and whether or not tick populations can be sustained or become established.

Ticks over-winter and rest while seeking hosts during warmer seasons in vegetation litter. Since ticks require sufficient humidity for survival, vegetation litter serves as a refuge from heat and dehydration. Ogden et al. (2006a) compared tick survival at several sites in south-eastern Canada that differed by vegetation type. The results suggested that this tick species will be able to establish itself in some new types of habitats, provided that there is a suitable density of hosts for the ticks (i.e., rodents, and deer). In addition, a theoretical investigation of the potential for ticks to expand their range in response to climate change (Ogden et al. 2006b) found that a northward range expansion was likely to be evident by 2020. One uncertainty in this modeling exercise is that rainfall was not included in the model, and this factor affects whether or not humidity and available moisture are sufficient to support the life cycle of ticks. In addition, some climate change scenarios suggest changes in rainfall pattern toward alternating periods of drought and heavy rainfall, which would likely reduce tick survival and range expansion.

Brownstein et al. (2003) developed a model to predict areas that could support established populations of *Ixodes scapularis* in the United States based on habitat suitability. The results showed that maximum, minimum and mean temperatures, along with vapor pressure all significantly contributed to tick population maintenance, although not all areas that were suitable based on habitat alone had resident tick populations. The resulting probability map was linked to a climate change model to estimate areas that will likely become suitable habitat areas for this species of tick in the future as climate warms. The model predicted that much of the United States will become potentially suitable habitat for this species of tick. It remains important to remember, however, that other factors, including availability of a suitable host species are required to support a tick population.

A study of the relationship between precipitation and occurrence of Lyme disease in the north eastern U.S. found that late spring/early summer precipitation was a significant climate-related factor affecting occurrence of Lyme disease (McCabe and Bunnell 2004). Ostfeld et al. (2006) found that the strongest predictors of current year risk of Lyme disease were the previous year's abundance of mice and chipmunks, and the abundance of acorns two years previously. Inclusion of meteorological factors or deer did not improve the predictions. These results

suggest that risk is related to prior abundance of hosts for the immature stages of the tick vector, and to availability of food for these hosts. Significant correlations between Lyme disease incidence and June moisture index two years previously and warmer winter weather a year and a half previous have been reported for the northeastern U.S. (Subak 2003). These factors may lead to higher survival of the important vector host, the white-footed mouse.

3.5.5. Rodent-Borne Diseases

Rodent-borne diseases are usually transmitted to humans through direct contact with rodent urine, feces or other body fluids. The most well-known rodent-borne human disease in the Southwest United States is hanta virus, although rodents are also hosts for fleas and ticks that can spread plague and several other infectious diseases (McMichael and Githeko 2001). The potential for human infection with hanta virus appears to be primarily related to the size of the rodent population. The impact of climate change on rodent-borne diseases is likely to be related to relatively short cycles, for example a year or two rather than longer-term, and based primarily on the availability of food, and consequently the rodent population (Gubler et al. 2001). There have been 49 cases of hanta virus cardiopulmonary syndrome in California since 1993 (CDPH 2008).

3.5.6. Conclusions

- Incidence of water-borne infectious diseases is influenced by the amount of precipitation, and storm intensity. These diseases are associated with contaminated run-off due to heavy precipitation and subsequent flooding and sewage overflow.
- The influence of climate and meteorology on vector-borne diseases is multifactorial. There are multiple points in the life cycles of the vector, the infectious agent, and host species that are potentially sensitive to local meteorology, and integration of the responses of each factor to climate change will determine the extent to which the ranges of these diseases alter.

3.6. Climate Change and Public Health

3.6.1. Future Estimates of Heat-Related Mortality and Morbidity

While numerous studies project that extreme heat conditions will increase with climate change in the future, and conjecture that this will increase the risk to human health and likely heat-related mortality (e.g., Karl et al., 2009), there are very few published studies that have attempted to make quantitative estimates of the likely future effects of increasing temperature on heat mortality. Two of these were done in California. The first, Hayhoe et al. (2004; Union of Concerned Scientists 2004) found that several different general circulation models employing several emissions scenarios pointed to the conclusion that there will likely be more hot days in the future, although not necessarily hotter temperatures on each day, under each scenario, along with an increase in the number and intensity of heat waves.

The second analysis, a part of the 2006 scenarios analysis project (Drechsler et al. 2006) focused on five California cities (Los Angeles, San Francisco, Fresno, San Bernardino, and Sacramento). The analysis concluded that climate change will likely result in a significant increase in the number of days with temperatures exceeding the ninetieth percentile of the historical temperature distribution (T90 day). All emissions scenarios (A1fi-high emissions, A2-medium-high emissions, and B1-lower emissions; IPCC 2007) and climate models (HadCM3, GFDL2.1,

and PCM)[1] point to longer, more intense, and more frequent heat waves, with the magnitude of the changes correlated with emissions; that is, scenarios based on higher greenhouse gas emissions predict greater increases than those with lower emissions, as illustrated in Figure 1 (Drechsler et al. 2006). By mid-century (2035–2064), temperatures currently seen on only 10% (or ~36 days) of the year will be exceeded on average 1.5 to 2.5 times more frequently each year. By the end of the century, extreme temperatures will be exceeded up to 4 times their current frequency under the high emissions scenario.

The frequency, length, and intensity of individual heat waves, as well as the duration of the entire heat wave season, are also projected to increase for all five cities studied. Inter-scenario differences are evident by mid-century, with significantly greater increases by century's end under the higher emissions (A1fi/A2) scenarios and for the more sensitive climate change models (GFDL and HadCM3). Results indicate large increases in the projected heat intensity, as compared to historical averages.

The analysis suggests that heat-related mortality with the high emissions scenarios (A1fi/A2) is likely to be almost double that for the lower emissions scenario (B1) by the end of the century. Unacclimatized mid-century projections show increases of 1.5 to 3 times the 1971–2000 values under B1 and 2 to 4.5 times under A1fi/A2, while end-of-century increases range from 2 to 4 times the historical values for each city for B1 and 3.5 to 9.5 times for A1fi/A2. The greatest overall increase in the heat-related mortality rate (per 100,000 people) is projected for Los Angeles. Since it is also the city with the largest population it is also projected to experience the greatest absolute impact as well. Overall, acclimatization is most successful at reducing projected mortality rates for inland cities that already experience extended periods of extreme heat and smallest for coastal cities where cool air masses off the ocean prevent consistent acclimatization to hotter conditions. For example, acclimatization reduced the mortality rates projected for Sacramento by 40% and for Fresno by 75%.

The only other U.S. study that has estimated heat-related mortality related to future climate change focuses on the New York metropolitan area. Knowlton et al. (2007) investigated the impact of projected regional increases in heat-related premature mortality to the year 2050. The results suggest an increase between 47% and 95% compared to the 1990s, without adaptations. The magnitude of the increase varied considerably across the region, and it was greater in urban counties than in less-urbanized counties. Inclusion of adaptations in the models reduced regional increases in summer heat-related premature mortality by about 25%.

It is important to note that demographic changes, societal decisions affecting implementation of adaptation measures, and changes in the health care sector will determine actual mortality rates. However, model uncertainties notwithstanding, extreme heat and associated human health risks under the lower-emissions scenario are significantly less than under higher-emissions scenarios. It is also important to note that increases in summer mean temperatures and the frequency, intensity, and duration of extreme heat events have significant implications for energy demand, particularly for additional electricity needed to support increased penetration of air conditioning in parts of the state where it is currently uncommon.

[1] The Hadley Centre Coupled Model, version 3; Geophysical Fluid Dynamics Laboratory model, version 2.1; and Parallel Climate Model.

There have been no projections of future morbidity related to increasing ambient temperatures related to climate change for any area of the United States.

3.6.2. Future Estimates of Cold-Related Mortality and Morbidity

Similar to the generic statements about heat-related health risks, general conjectures about declining cold-related diseases related to projections of increasing winter temperatures abound, but the knowledge base from research directly examining future cold-related mortality is slim indeed. There have been no projections of cold-related mortality or morbidity related to the projected shift in the range of ambient temperatures related to future climate change in the U.S.

3.6.3. Implications of Increasing Ambient Temperature on Air Quality

Rising temperature will increase the rate at which atmospheric chemical reactions proceed, increasing concentrations of ozone and possibly PM. However, relative humidity, wind speed, and mixing height also interact with temperature to affect the resulting pollutant concentrations (Drechsler et al. 2006; Steiner et al., 2006; Kleeman 2008; Mahmud et al. 2008; Jacobson, 2008; Millstein and Harley, 2009). These analyses also indicate that changes in the concentrations of ozone and PM are unlikely to be uniform across an air basin, making a health impacts analysis based on a simple incremental approach inappropriate. To date, air quality projections for California are limited to investigating the influence of perturbation of meteorological parameters on data from a few high air pollution episodes in the South Coast Air Basin and the San Joaquin Valley. These episodes lasted only a few days. While these analyses have provided important information as to the influence of meteorological parameters on air quality, the data are insufficient for projecting future health impacts.

Only one paper has attempted to quantitatively estimate the impact of future increases in ozone concentrations on mortality, and none have been published on PM.. Knowlton et al. (2004) estimated a 4.5% increase in ozone-related mortality for the summer season in the New York metropolitan area based on modeled regional ozone concentrations using projected ozone precursor emissions with several climate change scenarios. The models, however, did not investigate whether ozone concentrations confounded or modified the temperature-mortality relationship. However, modeling studies investigating the influence of climate change on ozone concentrations suggest that without implementation of new ozone control measures mortality related to ozone could increase in the future, while the influence of climate change on future PM concentrations is less certain (Millstein & Harley, 2009; Drechsler et al. 2006).

California is taking steps toward attaining the existing ambient air quality standards for ozone and PM. Although limited, the information available on the influence of climate change on ozone and PM makes it clear that increasing temperature will make attainment of these standards more difficult in the future (Millstein & Harley, 2009; Drechsler et al. 2006). Since health impacts attributable to air pollution are related to ambient concentrations, the magnitude of health impacts attributable to ozone and PM in the future will be proportional to the degree to which ambient air quality standards are not attained.

3.6.4. Implications of increasing Ambient Temperatures on Wildfires

Wildfires can be a significant contributor to air pollution in both urban and rural areas, and they have the potential to significantly affect public health primarily through their smoke. Fires also affect the economy and public safety. Various climate change scenarios project that through the

twenty-first century there will likely be an increase in the frequency, size, and intensity of wildfires (Westerling et al. 2009). However, quantitative estimation of the public health impacts of future wildfire events is extremely difficult for several reasons. The public health impacts of any fire are unique to that fire, and are influenced not only by the magnitude, intensity, and duration of the fire, but also the proximity of the smoke plume to a population. Predictive assessments are further complicated by the fact that there are usually few, if any, air quality data available during fires other than those from the ambient air quality monitoring network, which are often not representative of air quality in the fire-impacted area. In addition, population densities in exposed communities are rarely large enough to yield sufficient sample sizes for assessment of epidemiologic relationships between poor air quality due to fires and health endpoints.

3.6.5. Implications of increasing Ambient Temperature on Infectious Diseases

Meteorological changes can influence human disease through both direct and indirect effects on pathologic microorganisms, vectors, reservoirs, and hosts (Colwell and Patz 1998). Available evidence suggests that the incidence and spread of a number of infectious diseases can be affected by various weather-related factors on a time scale of a few years, and that climate change has the potential to affect their range, incidence and spread (Colwell and Patz 1998).

Most research to date on the impacts of climate change on infectious diseases has focused on short-term changes in weather patterns, primarily in rainfall, humidity, and ambient temperature, as opposed to long-term changes related to global climate change, largely because of the significant influence of relatively short-term weather patterns of the ecology and range of pathogens. The interactions between host and infectious organisms are complex, and the impact of climate on the ecology of infectious diseases increases that complexity, making it difficult to predict changes that may result from climate change. However, on a global scale, there is concern that rising temperatures may encourage the growth of infectious organisms, and thus increase global disease burden (Rose et al. 2001). Global travel may also increase the risk of introducing infectious diseases that are not currently endemic to California.

A National Research Council report (2001) that reviewed the relationships among climate, ecosystems and infectious diseases made several conclusions, including:

- Changes in weather, in both the short and longer terms can influence infectious diseases.
- Observational and modeling studies must be interpreted cautiously because they are unable to fully account for all of the factors that influence infectious diseases.
- Climate change may alter the evolution and ranges for infectious diseases.
- Since infectious diseases are highly dependent on local-scale and comparatively short-term weather parameters, extrapolation of climate and disease relationships from one spatial or temporal scale to another is not likely to yield valid estimates of future impacts.

3.6.6. Water- and Food-Borne Diseases

It is not possible to predict the likely incidence of diseases related to contaminated water or food, due to the complex interactions between the contamination potential of these pathogens, the effectiveness of public health programs designed to minimize exposure of the public to

contaminated water and food, and the public's compliance with these programs and recommendations.

Climate modeling suggests that extreme precipitation events, with significant runoff, are likely to become more commonplace as California's climate warms (Karl et al. 2008; Mastrandrea et al. 2009; Cayan et al. 2009; Dettinger et al. 2009). Coastal areas of California, where urban growth and development and land use decisions influence the quality of runoff water flowing through creeks and rivers to coastal beaches, are especially vulnerable to water contamination. This is particularly the case in the southern part of the state, where expanding urbanization, development and an increasing population generate pollution that ultimately flows into the Pacific Ocean (Dwight et al. 2002). Temporal and spatial analysis of bacterial levels near the mouths of several Los Angeles area rivers that empty into the Pacific Ocean showed that bacteria levels were highest near river mouths and adjacent beaches, and that precipitation events were significantly associated with increased bacterial loads in the same areas (Dwight et al. 2002). Louis et al. (2003) reported similar findings for the Chesapeake Bay on the East Coast of the United States. Severe precipitation events could increase opportunities for sewage treatment facilities to fail, leading to public exposure to contaminated water. Combined sewer systems, which carry both storm water and raw sewage to treatment plants, are a significant source of drinking and recreational water contamination, and although they are considered antiquated, some communities in California continue to be served by these systems. When overtaxed, these systems overflow directly into a surface water body, for example a river or lake, introducing untreated storm runoff water and sewage, along with any pathogens, pollutants, chemicals, and industrial wastes they contain, directly into the water body, thereby contaminating the water body, beaches, fish, and shellfish (Charron et al. 2004).

In the coastal zone, toxic algal blooms will likely be more frequent as water temperature rises, increasing the risk of illness originating from aquatic recreation, such as swimming and surfing, and from contamination of seafood (Rose et al. 2001). Food supplies could also become contaminated through contaminated runoff and through lack of field sanitation that results in contaminated irrigation water (Rose et al. 2001).

Food-borne diseases can originate with contamination of fruits, vegetables, and seafood by flooding, contaminated runoff following heavy rain, contaminated irrigation water, or for seafood by toxic substances released by algal blooms (Rose et al. 2001). The extent to which these diseases will increase is affected by multiple factors. It is likely that irrigation water contamination due to flooding, runoff, or seepage from adjacent livestock operations could increase due to more rapid organism replication under future warming conditions (Lipp et al. 2002; Hunter 2003). In addition, more frequent toxic algal blooms may occur under conditions of increased water temperature, leading to increased incidences of seafood contamination, in addition to increased contamination with Vibrio species.

Although food can be contaminated in the fields, or during harvest or post-harvest processing, most outbreaks of food-borne diseases are related to improper food handling or storage, either at home, in restaurants, or at food stores. Food-borne illnesses could increase due to more rapid bacterial proliferation at warmer temperatures in food not handled correctly during harvesting or processing, or not maintained at proper storage temperatures (Colwell and Patz 1998). A study done in England by Kovats et al. (2004b,c) found that increasing ambient temperature contributed to transmission of salmonella and campylobacter through food.

Those most at risk of experiencing serious effects from water- and food-borne diseases are the very young, the elderly, the infirm, and people with compromised immune systems. The California Department of Public Health and California counties and cities conduct surveillance for these diseases, as well as having protocols that are activated when outbreaks of disease occur. However, surveillance for these diseases is relatively poor, since most cases do not require or seek medical attention, leading to underreporting.

3.6.7. Other Infectious Diseases

Few studies have investigated the influence of weather factors on infectious diseases spread through oral-fecal or respiratory routes, such as enteritis and influenza. However, several studies suggest that increasing temperature and the ENSO could influence incidence of these diseases, although there are insufficient data available to estimate future cases of disease.

3.6.8. Vector-Borne Diseases

Vector-borne diseases are caused by a variety of viruses, bacteria, and protozoa that spend part of their life cycle in a host species (mosquitoes, fleas, and ticks) and are spread to humans and animals during insect feeding. The literature is clear that disease transmission rates are not only related to the complex interactions between factors related to vector and infectious organism survival, but also to other factors that indirectly affect the vector and organism. Such factors include the availability of suitable habitat for vector growth, reproduction, and maturation; land use patterns; vector control programs; and personal behaviors—including use of window and door screens, insecticides and repellents, clothing selection, availability of preventative vaccines and drugs to treat active cases, and being outdoors when the vector is active (Kovats et al. 2001; Sutherst 2004).

Both ticks and mosquitoes and the viruses they can spread are temperature sensitive, although the transmission and maturation of these viruses is dependent on many factors in addition to ambient temperature, such as availability of a suitable habitat. Each of these viruses has a different pattern of susceptibility to climate factors, including temperature, humidity, and rainfall.

Natively acquired cases of other mosquito-borne diseases such as malaria and dengue fever, which require no animal host, are extremely rare in the United States (Gubler et al. 2001), although malaria was once endemic to most of the country. However, predictions of increased numbers of people globally living in areas where malaria is endemic, coupled with increased world travel, increases the possibility of additional cases of malaria and other currently non-endemic diseases in California (Pherez 2007).

It is likely that the ranges for these infectious diseases will shift (Gubler et al. 2001) in response to global warming. However, modeling to date does not generally take into account the large number of factors unrelated to climate that also influence vectors, infectious organisms, and disease transmission rates. These factors include land use patterns, availability of suitable habitats, availability of suitable hosts, and natural and artificial barriers to species dispersal (Kovats et al. 2001; Pherez 2007). Analysis of the history of three vector-borne diseases (malaria, yellow fever, and dengue fever) not currently endemic in the United States (Reiter 2001) suggests that climate, at least on the order of several years, has rarely been the principal determinant of the prevalence or range of these diseases. Rather, the most important determinants have been human activities and their impact on local ecology, suggesting that the

use of climate modeling to estimate future vector-borne disease rates from likely vector ranges may be inappropriate.

Several studies have emphasized the human behavior aspect of these diseases. Randolph et al. (2008) found that there were significant differences in tick-borne encephalitis rates among European countries that were more related to human behavior, including extent of outdoor activity in tick-infested regions, than to tick abundance and activity. Although focused on dengue virus infection, which is not endemic in California, results by Reiter et al. (2003) are illustrative of the influence of human behavior on transmission of vector-borne diseases. Laredo, Texas and Nuevo Laredo, Mexico are separated only by the Rio Grande River. Although there was greater abundance of the mosquito vector that carries dengue virus in Laredo, the disease rate was higher in Nuevo Laredo. Human behavior and environmental factors that influence contact with mosquitoes appear to account for this paradox. Houses in Laredo were more likely to have window screens and air conditioning, and people were less likely to sit outside in the evenings, compared to in Nuevo Laredo, suggesting that human exposure to the vector is an important factor in disease transmission rate.

The people most at risk for serious effects from these diseases are the very young, the elderly, the infirm, and people with compromised immune systems. Most healthy people are at little risk of serious disease from infection with these organisms (California Department of Health Services 2004). Exposure to these organisms leads to antibody formation that persists for at least several years; however, the degree and duration of protection provided by antibodies from an initial infection is unknown because these diseases are rare enough that there are no data available on reinfection rates (Personal communication, California Department of Public Health).

California has well-developed protocols involving the State, counties, and cities for surveillance for these diseases. The State also has a multi-level protocol that is activated based on risk of disease transmission (CDPH 2004; Barker et al. 2003). It is possible that the small number of cases of these diseases in California, compared to the large populations living in endemic areas, is related to the effectiveness of these and similar programs and recommendations such as use of window screens, insect repellants, and not going outdoors during times of high mosquito activity (Barker et al. 2003).

3.6.9. Rodent-Borne Diseases

Rodent-borne diseases are usually transmitted to humans through direct contact with rodent urine, feces or other body fluids. The most well-known rodent-borne human disease in the Southwest United States is hanta virus, although rodents are also hosts for fleas and ticks that can spread plague and several other infectious diseases (McMichael and Githeko 2001). The potential for human infection with hanta virus appears to be primarily related to the size of the rodent population. The impact of climate change on rodent-borne diseases is likely to be related to relatively short cycles, for example a year or two rather than longer-term, and based primarily on the availability of food, and consequently the rodent population (Gubler et al. 2001). There have been 49 cases of hanta virus cardiopulmonary syndrome in California since 1993 (CDPH 2008).

4.0 Conclusions and Recommendations

4.1. Conclusions

4.1.1. Temperature-Related Mortality and Morbidity

The temperatures at which heat and cold-related mortality occur vary among locations, and are primarily related to deviations from local average temperatures. Consequently, temperature deviations both above and below the typical levels are associated with an increased risk of temperature-related mortality for both excess deaths, and for deaths directly caused by heat or cold. This means that temperature alert programs must be tailored to specific regions, rather than application of a "one-size fits all" approach.

The majority of excess deaths related to heat appear to occur in cities, pointing to the influence of the heat island effect in urban areas, where temperatures are several degrees higher than in less densely populated areas outside of central city areas.

Most excess heat-related deaths occur in older people, particularly over age 75, and more frequently in women than men. There is also increased risk of mortality directly attributable to heat for younger people who are active outdoors in the heat, particularly outdoor workers. Identified risk factors for heat-related mortality include being bed-ridden or otherwise having an increased level of dependency, not leaving home daily, living alone, living of the top floor of a building, having few social contacts, lack of access to transportation, and lack of access to or not using air conditioning. A number of common clinical conditions have been associated with increased risk of heat mortality, including cardiovascular, respiratory, neurological, and psychiatric diseases. In addition, a number of common medications used to treat these diseases adversely affect the body's ability to thermoregulate, and increase risk of heat-related mortality.

Analyses in Chicago, Milwaukee, Philadelphia, Boston, and other cities worldwide have demonstrated that heat health watch, warning, and emergency action plans are effective is reducing heat-related morbidity and mortality.

Populations accustomed to cold climates typically experience less cold-related mortality than areas with milder climates, and many cold-related deaths occur at relatively mild temperatures. This counterintuitive finding suggests that people living in areas with mild winters may lack appreciation of the dangers of cold, may have insufficient home heating and insulation, or fail to wear adequate clothing for the ambient conditions. As California warms, there could be a decrease in cold-mortality, but it would be a mistake to conclude that cold temperatures no longer will pose a risk. People tend to be unprepared for conditions outside the typical. Failure to remain cognizant of the public health risks associated with cold temperatures will continue to put the elderly, the poor, and the homeless at increased risk of cold-related mortality, even if the episode is relatively mild or lasts for only a few days. Awareness of the behavioral aspect of people's response to heat and cold may also aid in modifying existing watch/warning systems and provide appropriate reminders to individuals from official sources.

4.1.2. Air Pollution

Air pollution-related health effects are fundamentally tied to the extent to which ambient air quality standards are not attained. The influence of increasing temperatures on both ambient and global background concentrations of air pollutants will likely increase the difficulty of

attaining current ambient air quality standards for ozone (and possibly for PM10 and PM2.5), and increase the cost and time period required to reach attainment. It is also likely that as temperatures increase, people will alter their behavior and activity patterns, resulting in increased time indoors, which would tend to reduce exposure, particularly as air conditioning becomes increasingly prevalent. Air conditioning, which is projected to become increasingly prevalent statewide, typically reduces the concentrations of ozone and PM indoors compared to outdoors. Indoor ozone levels typically range between 20% and 80% of outdoor levels, depending on such factors as season, building ventilation rate and microenvironmental factors (Weschler et al. 1989). Consequently, if global warming causes people to increase the amount of time they spend indoors in air conditioned environments, ozone- and PM-related health effects could, in theory, decrease, even if outdoor concentrations increase. However, it is not reasonable to assume that people will remain indoors at all times as California's climate warms. Also, the indoor environment can contain toxic air contaminants at concentrations that are several-fold higher than observed in outdoor ambient air. In addition, it should not be forgotten that there will continue to be a significant population of outdoor workers (e.g., gardeners, construction, and agricultural workers), as well as children and adults engaged in outdoor activities, who will continue to be at risk of adverse health outcomes related to ambient concentrations of air pollutants.

4.1.3. Wildfires

Review of the literature on wildfires indicates that fire smoke exposure can increase adverse respiratory health effects, including hospitalization, emergency room visits, and respiratory symptoms. The number of people who will be affected by future fires cannot be estimated due to the unique nature of each fire, and the necessity that the smoke plumes cover an inhabited area. However, the literature suggests that the people most at risk will be those with existing cardiopulmonary disease, and that risk increases with advancing age. Published analyses of morbidity and mortality related to wildfires suggest that the number and severity of additional cases related to future wildfires is likely to be modest.

The California Thoracic Society (1997), and the California Air Resources Board (CARB 2008) have published fire fact sheets that describe personal actions that can minimize lung injury when exposed to a fire. In addition, the document "Wildfire Smoke: A Guide for Public Health Officials (available at www.arb.ca.gov/smp/progdev/pubeduc/wfgv8.pdf) describes health risks related to fire smoke inhalation and actions that can be taken to protect oneself. It also provides guidance to public health officials on assessing the level of public health risk a particular fire poses, and recommended responses.

4.1.4. Infectious Diseases

A variety of infectious diseases could increase with climate change, although few data are available that address this question directly. These include diseases such as cryptosporidiosis or salmonellosis, which are contracted respectively through contaminated water or food, and those spread by various vectors, such as mosquitoes, ticks, and rodents, and include West Nile virus, Lyme disease, and Hanta virus. The local, State, and federal governments have active surveillance protocols in place to identify outbreaks of these diseases. Maintaining and strengthening California's public health infrastructure, along with active surveillance for water-, vector-, and food-borne diseases, are critical to preventing increased disease. Those most at risk

of experiencing serious effects from these infectious diseases are the very young, the elderly, the infirm, and people with compromised immune systems.

California also has an extensive protocol for combating vector-borne diseases. There are no human vaccines for these diseases, and consequently mitigations rely heavily on disease surveillance and vector control programs, as well as rapid and effective response once a disease has been contracted. A variety of personal behaviors, such as use of window screens and air conditioning, along with public health advisories to wear clothing that provides skin coverage, use insect repellent, and remain indoors during heightened vector activity times (for example, at dusk) are effective in reducing risk of contracting these diseases. Drainage of marshy areas, unmaintained swimming pools, and other sources of standing water, such as old tires, cans, and flower pots are also effective means of reducing habitat for mosquitoes, and thus the number of insects. Other possible habitats for mosquitoes include water storage, and distribution systems related to irrigation

4.2. Recommendations

4.2.1. Actions to Protect Public Health

The following are recommended actions that can help alleviate the impacts of climate change on public health.

- Strengthen surveillance for temperature-related mortality and adverse health effects of air pollution exposure and wildfires, as well as infectious diseases related to water-, vector-, and food-borne pathogens.
- Expand public education on the risks of extreme temperatures, high air pollution, wildfires, and infectious diseases related to water-, vector-, and food-borne pathogens to individuals and their caregivers. There should be an emphasis on effective steps that the individual can adopt to protect him- or herself. Educational materials should also cover personal risk factors for adverse outcomes, including age, health status, and commonly used prescription medications. Educational materials should be available in multiple languages to reach wider audiences.
- Improve coordination among alert systems and emergency personnel responding to public health emergencies related to temperature, air pollution, infectious diseases, and wildfires.
- Develop Health Heat Watch Warning Systems for regions of the State that have not yet adopted them. To date, only the San Jose area has a Health Heat Watch Warning System in place. These systems should be coupled with community-level programs to provide outreach and services to people in need of cooling centers and other assistance to prevent heat-related illness or death. These programs should focus primarily on the elderly and infirm but should not overlook the economically disadvantaged.
- Review, strengthen, and enforce occupational safety standards to protect outdoor workers from heat illness and mortality.
- Increase access to air conditioning, particularly in areas where it is currently not common and in population groups that lack access. Consider expanding existing programs that help low-income people pay for residential heating to include residential cooling as well.

- Evaluate the capacity of statewide water and sewage treatment facilities, and modernize and expand these facilities as necessary to meet predicted scenarios of extreme precipitation and runoff events. Replace remaining combined sewage systems in the State with modern systems.
- Review and expand existing vector control programs as necessary.
- Consider climate change as part of planning efforts directed at attaining the health-based ambient air quality standards.
- Encourage individuals and families to have an evacuation plan in the event of wildfire, keeping in mind that a wildfire may disrupt usual travel routes. These plans should include provisions for sheltering in place if not directed by authorities to evacuate, and should include planning for food and especially water in the event that utility service is disrupted by the fire.

4.2.2. Research Needs

This review suggests several avenues for future research.

- The health-related endpoints discussed above are influenced by many factors. Climate is one contributor, but these health endpoints are also influenced by many other factors unrelated to climate, including personal behaviors. Consequently, the ultimate health outcome is the result of the integration of all of these factors. Increasingly sophisticated statistical models will be required to make credible estimates of future climate change-related health impacts. These models will need to account for changes in population, healthcare, the physical environment (i.e., land use, building stock characteristics), life style factors, concurrent non-climatic stressors, and introduction of climate change adaptation strategies. The interactions among these various factors are unlikely to be simply additive, but synergistic, thus requiring sophisticated modeling.
- Research into the effects of climate change on air quality is still in its infancy, and results to date are primarily based on perturbations of single meteorological parameters within short, historical high air pollution episodes. These methods need to be expanded to better investigate the influence of projected complex meteorological changes on air pollution. These results will help to inform and guide development of new control technologies and regulations aimed at attaining the health-based ambient air quality standards.
- While there has been quite a bit of research into the short-term influence of meteorological factors on infectious diseases, for example as a means to predict incidence in the next season, little is known about the longer-term influence of climate-related factors on these diseases, particularly when vector control programs are factored into the analysis.
- Since most, if not all of the climate-related health effects discussed above are preventable with appropriate action on the part of health care providers, emergency responders, communities, individuals and their caregivers, a particularly fruitful avenue for future research would be development of more effective public health education and intervention strategies to equip communities, health care providers, and citizens to best protect themselves from weather and climate-related health impacts. This area of

research should also include investigations into improved methods for effecting behavior modifications that reduce health-related impacts of climate change.

- Significantly more research is needed into the potential, costs, and effectiveness of various adaptation strategies and their feasibility in different community contexts, including how to communicate these strategies most effectively.

5.0 References

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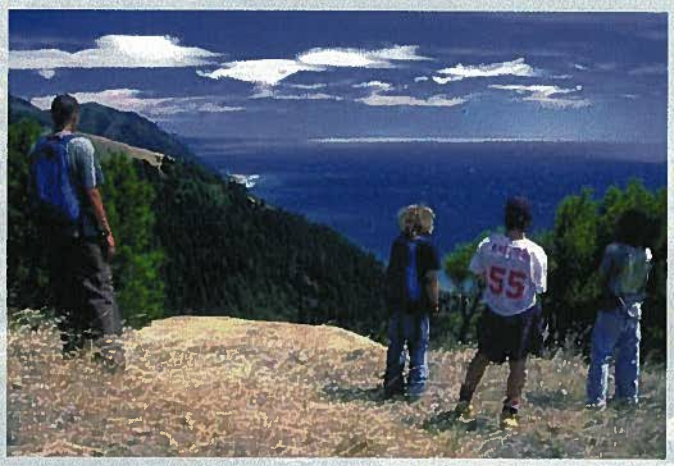
Our Changing Climate

Assessing the Risks to California

A Summary Report from
the California Climate Change Center

Because most global warming emissions remain in the atmosphere for decades or centuries, the choices we make today greatly influence the climate our children and grandchildren inherit. The quality of life they experience will depend on if and how rapidly California and the rest of the world reduce these emissions.

In California and throughout western North America, signs of a changing climate are evident. During the last 50 years, winter and spring temperatures have been warmer, spring snow levels in lower- and mid-elevation mountains have dropped, snowpack has been melting one to four weeks earlier, and flowers are blooming one to two weeks earlier.



These regional changes are consistent with global trends. During the past 100 years, average temperatures have risen more than one degree Fahrenheit worldwide. Research indicates that much of this warming is due to human activities, primarily burning fossil fuels and clearing forests, that release carbon dioxide (CO_2) and other gases into the atmosphere, trapping in heat that would otherwise escape into space. Once in the atmosphere, these heat-trapping emissions remain there for many years—CO_2, for example, lasts about 100 years. As a result, atmospheric concentration of CO_2 has increased more than 30 percent above pre-industrial levels. If left unchecked, by the end of the century CO_2 concentrations could reach levels three times higher than pre-industrial times, leading to dangerous global warming that threatens our public health, economy, and environment.

The latest projections, based on state-of-the art climate models, indicate that if global heat-trapping emissions proceed at a medium to high rate, temperatures in California are expected to rise 4.7 to 10.5°F by the end of the century. In contrast, a lower emissions rate would keep the projected warming to 3 to 5.6°F. These temperature increases would have widespread consequences including substantial loss of snowpack, increased risk of large wildfires, and reductions in the quality and quantity of certain agricultural products. The state's vital resources and natural landscapes are already under increasing stress due to California's rapidly growing population, which is expected to grow from 35 million today to 55 million by 2050.

This document summarizes the recent findings of the California Climate Change Center's "Climate Scenarios" project, which analyzed a range of impacts that projected rising temperatures would likely have on California. The growing severity of the consequences as temperature rises underscores the importance of reducing emissions to minimize further warming. At the same time, it is essential to identify those consequences that may be unavoidable, for which we will need to develop coping and adaptation strategies.

In 2003, the California Energy Commission's Public Interest Energy Research (PIER) program established the California Climate Change Center to conduct climate change research relevant to the state. This Center is a virtual organization with core research activities at Scripps Institution of Oceanography and the University of California, Berkeley, complemented by efforts at other research institutions. Priority research areas defined in PIER's five-year Climate Change Research Plan are: monitoring, analysis, and modeling of climate; analysis of options to reduce greenhouse gas emissions; assessment of physical impacts and of adaptation strategies; and analysis of the economic consequences of both climate change impacts as well as the efforts designed to reduce emissions.

Executive Order #S-3-05, signed by Governor Arnold Schwarzenegger on June 1, 2005, called for the California Environmental Protection Agency (CalEPA) to prepare biennial science reports on the potential impact of continued global warming on certain sectors of the California economy. CalEPA entrusted PIER and its California Climate Change Center to lead this effort. The "Climate Scenarios" analysis summarized here is the first of these biennial science reports, and is the product of a multi-institution collaboration among the California Air Resources Board, California Department of Water Resources, California Energy Commission, CalEPA, and the Union of Concerned Scientists.

Cover photos: (sunset) Photos.com; (from top to bottom) AP Photo/Paul Sakuma, IStockphoto, IndexStock, Picturequest, IStockphoto. Above: Bureau of Land Management. Background: IndexStock

California's Future Climate

California's climate is expected to become considerably warmer during this century. How much warmer depends on the rate at which human activities, such as the burning of fossil fuels, continue. The projections presented here illustrate the climatic changes that are likely from three different heat-trapping emissions scenarios (see figure below).

Projected Warming

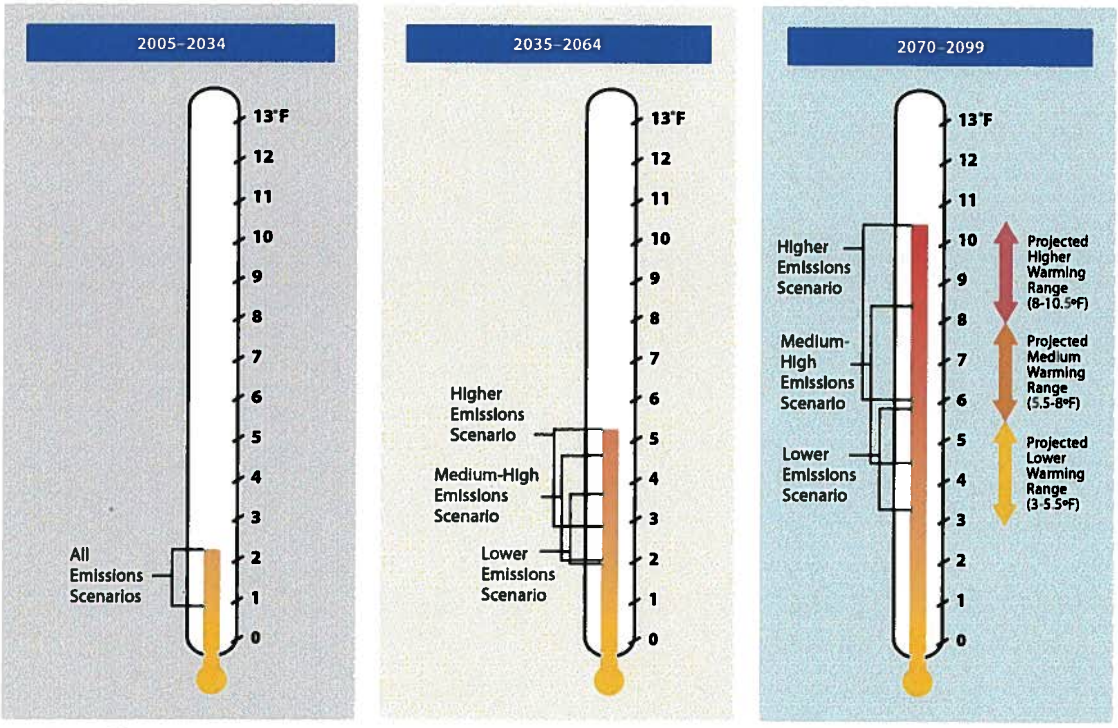
Temperatures are expected to rise substantially in all three emissions scenarios. During the next few decades, the three scenarios project average temperatures to rise between 1 and 2.3°F; however, the projected temperature increases begin to diverge at mid-century so that, by the end of the century, the temperature increases projected in the higher emissions scenario are approximately twice as high as those projected in the lower emissions scenario. Some climate models indicate that warming would be greater in summer than in winter, which would have widespread effects on ecosystem health, agricultural production, water use and availability, and energy demand.

Toward the end of the century, depending on future heat-trapping emissions, statewide average temperatures are expected to rise between 3 and 10.5°F. The analysis presented here examines the future climate under three projected warming ranges:[1]

- **Lower warming range:** projected temperature rises between 3 and 5.5°F
- **Medium warming range:** projected temperature rises between 5.5 and 8°F
- **Higher warming range:** projected temperature rises between 8 and 10.5°F

Precipitation

On average, the projections show little change in total annual precipitation in California. Furthermore, among several models, precipitation projections do not show a consistent trend during the next century. The Mediterranean seasonal precipitation pattern is expected to continue, with most precipitation falling during winter from North Pacific storms. One of the three climate models projects slightly wetter winters, and another projects slightly drier winters with a 10 to 20 percent decrease in total annual precipitation. However, even modest changes would have a significant impact because California ecosystems are conditioned to historical precipitation levels and water resources are nearly fully utilized.



California is expected to experience dramatically warmer temperatures during the 21st century. This figure shows projected increases in statewide annual temperatures for three 30-year periods. Ranges for each emissions scenario represent results from state-of-the-art climate models.

1 These warming ranges are for illustrative purposes only. These ranges were defined in the original Climate Scenarios analysis to capture the full range of projected temperature rise. The exact values for the warming ranges as presented in the original summary report are: lower warming range (3 to 5.4°F); medium warming range (5.5 to 7.9°F); and higher warming range (8 to 10.4°F).

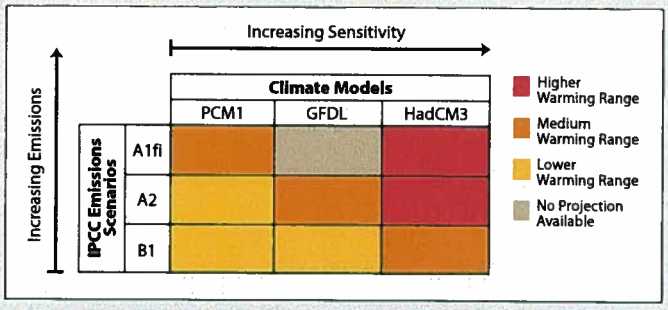
Projecting Future Climate

How much temperatures rise depends in large part on how much and how quickly heat-trapping emissions accumulate in the atmosphere and how the climate responds to these emissions. The projections presented in this report are based on three different heat-trapping emissions scenarios and three climate models.

Emissions Scenarios

The three global emissions scenarios used in this analysis were selected from a set of scenarios developed by the Intergovernmental Panel on Climate Change's (IPCC) *Special Report on Emissions Scenarios*, based on different assumptions about population growth and economic development (measured in gross domestic product).

- The **lower emissions scenario (B1)** characterizes a world with high economic growth and a global population that peaks by mid-century and then declines. There is a rapid shift toward less fossil fuel-intensive industries and introduction of clean and resource-efficient technologies. Heat-trapping emissions peak about mid-century and then decline; CO_2 concentration approximately doubles, relative to pre-industrial levels, by 2100.
- The **medium-high emissions scenario (A2)** projects continuous population growth and uneven economic and technological growth. The income gap between now-industrialized and developing parts of the world does not narrow. Heat-trapping emissions increase through the 21st century; atmospheric CO_2 concentration approximately triples, relative to pre-industrial levels, by 2100.
- The **higher emissions scenario (A1fi)** represents a world with high fossil fuel-intensive economic growth, and a global population that peaks mid-century then declines. New and more efficient technologies are introduced toward the end of the century. Heat-trapping emissions increase through the 21st century; CO_2 concentration more than triples, relative to pre-industrial levels, by 2100.



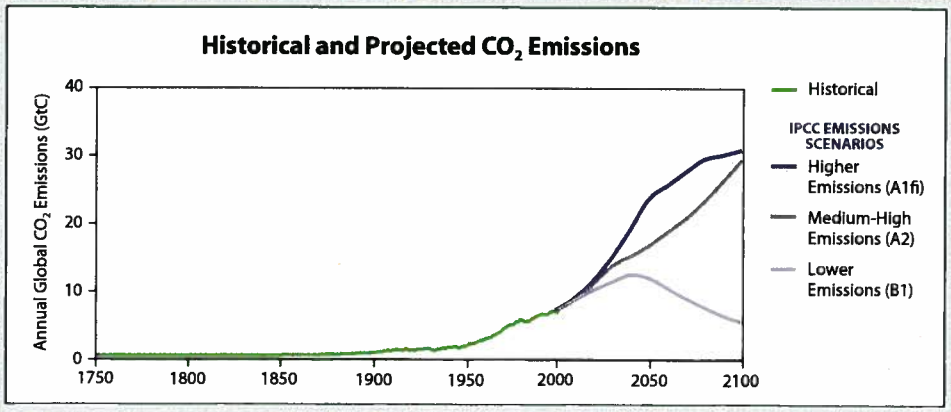
This matrix shows the temperature increases that result from the three climate models, assuming emission inputs indicated in the IPCC emissions scenarios. The resulting temperatures are grouped into three warming ranges defined in the "Climate Scenarios" analysis.

Climate Sensitivity

The three models used in this analysis represent different climate sensitivities, or the extent to which temperatures will rise as a result of increasing atmospheric concentrations of heat-trapping gases. Climate sensitivity depends on Earth's response to certain physical processes, including a number of "feedbacks" that might amplify or lessen warming. For example, as heat-trapping emissions cause temperatures to rise, the atmosphere can hold more water vapor, which traps heat and raises temperatures further—a positive feedback. Clouds created by this water vapor could absorb and re-radiate outgoing infrared radiation from Earth's surface (another positive feedback) or reflect more incoming shortwave radiation from the sun before it reaches Earth's surface (a negative feedback).

Because many of these processes and their feedbacks are not yet fully understood, they are represented somewhat differently in different global climate models. The three global climate models used in this analysis are:

- **National Center for Atmospheric Research Parallel Climate Model (PCM1):** low climate sensitivity
- **Geophysical Fluids Dynamic Laboratory (GFDL) CM2.1:** medium climate sensitivity
- **United Kingdom Met Office Hadley Centre Climate Model, version 3 (HadCM3):** medium-high climate sensitivity



As this figure shows, CO_2 emissions from human activities (such as the burning of fossil fuels) were negligible until around the so-called industrial age starting in the 1850s.

Public Health

Continued global warming will affect Californians' health by exacerbating air pollution, intensifying heat waves, and expanding the range of infectious diseases. The primary concern is not so much the change in *average* climate but the projected increase in *extreme* conditions, which pose the most serious health risks.

Poor Air Quality Made Worse

Californians currently experience the worst air quality in the nation, with more than 90 percent of the population living in areas that violate the state's air quality standard for either ground-level ozone or airborne particulate matter. These pollutants can cause or aggravate a wide range of health problems including asthma and other acute respiratory and cardiovascular diseases, and can decrease lung function in children. Combined, ozone and particulate matter contribute to 8,800 deaths and $71 billion in healthcare costs every year. If global background ozone levels increase as projected in some scenarios, it may become impossible to meet local air quality standards.

Higher temperatures are expected to increase the frequency, duration, and intensity of conditions conducive to air pollution formation. For example, if temperatures rise to the medium warming range, there will be 75 to 85 percent more days with weather conducive to ozone formation in Los Angeles and the San Joaquin Valley, relative to today's conditions. This is more than twice the increase expected if temperature rises are kept in the lower warming range.

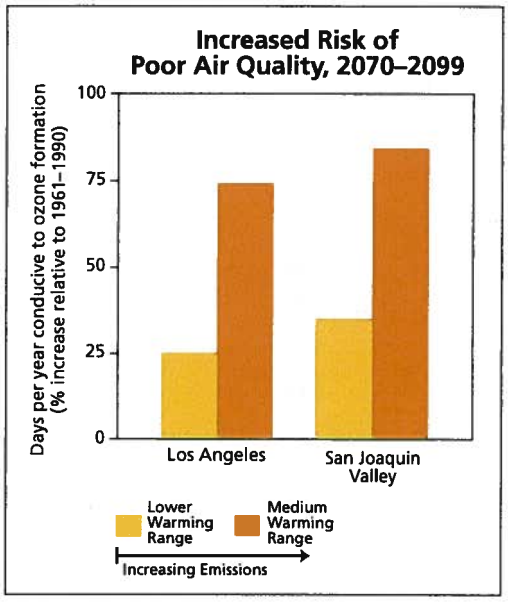
Air quality could be further compromised by increases in wildfires, which emit fine particulate matter that can travel long distances depending on wind conditions. The most recent analysis suggests that if heat-trapping gas emissions are not significantly reduced, large wildfires could become up to 55 percent more frequent toward the end of the century.

More Severe Heat

By 2100, if temperatures rise to the higher warming range, there could be up to 100 more days per year with temperatures above 90°F in Los Angeles and above 95°F in Sacramento. This is a striking increase over historical patterns (see chart on p. 6), and almost twice the increase projected if temperatures remain within or below the lower warming range.

As temperatures rise, Californians will face greater risk of death from dehydration, heat stroke/exhaustion, heart attack, stroke, and respiratory distress caused by extreme heat. By mid century, extreme heat events in urban centers such as Sacramento, Los Angeles, and San Bernardino could cause two to three times more heat-related deaths than occur today. The members of the population most vulnerable to the effects of extreme heat include people who are already ill; children; the elderly;

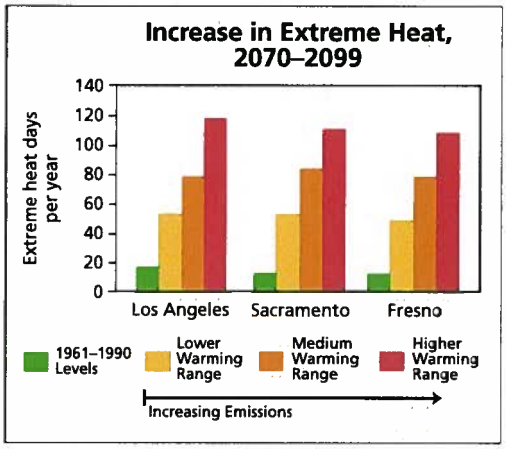
As temperatures rise, Californians will face greater risk of death from dehydration, heat stroke, heart attack, and other heat-related illnesses.

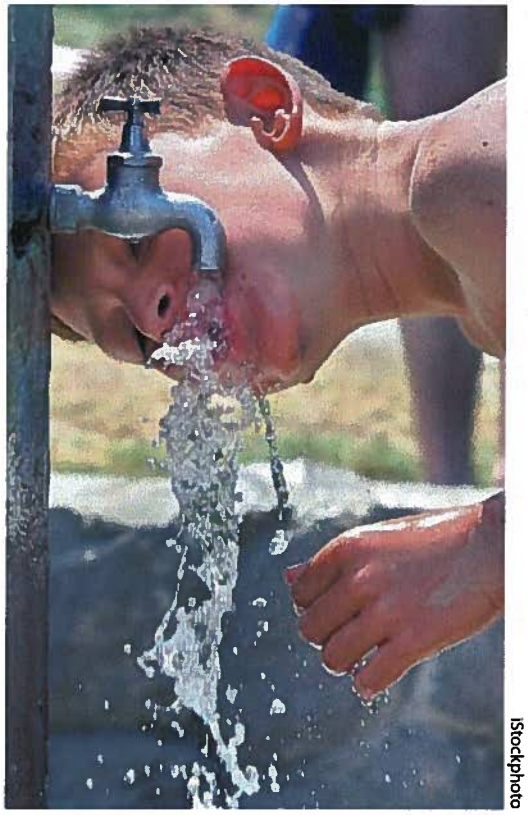


Cars and power plants emit pollutants that contribute to global warming and poor air quality. As temperatures increase, it will be increasingly difficult to meet air quality standards throughout the state.

and the poor, who may lack access to air conditioning and medical assistance.

More research is needed to better understand the potential effects of higher temperatures and the role that adaptation can play in minimizing these effects. For example, expanding air conditioner use can help people cope with extreme heat; however, it also increases energy consumption, which, using today's fossil fuel-heavy energy sources, would contribute to further global warming and air pollution.





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Water Resources

If global warming emissions continue unabated, Sierra Nevada snowpack could decline 70 to 90 percent, with cascading effects on winter recreation, water supply, and natural ecosystems.

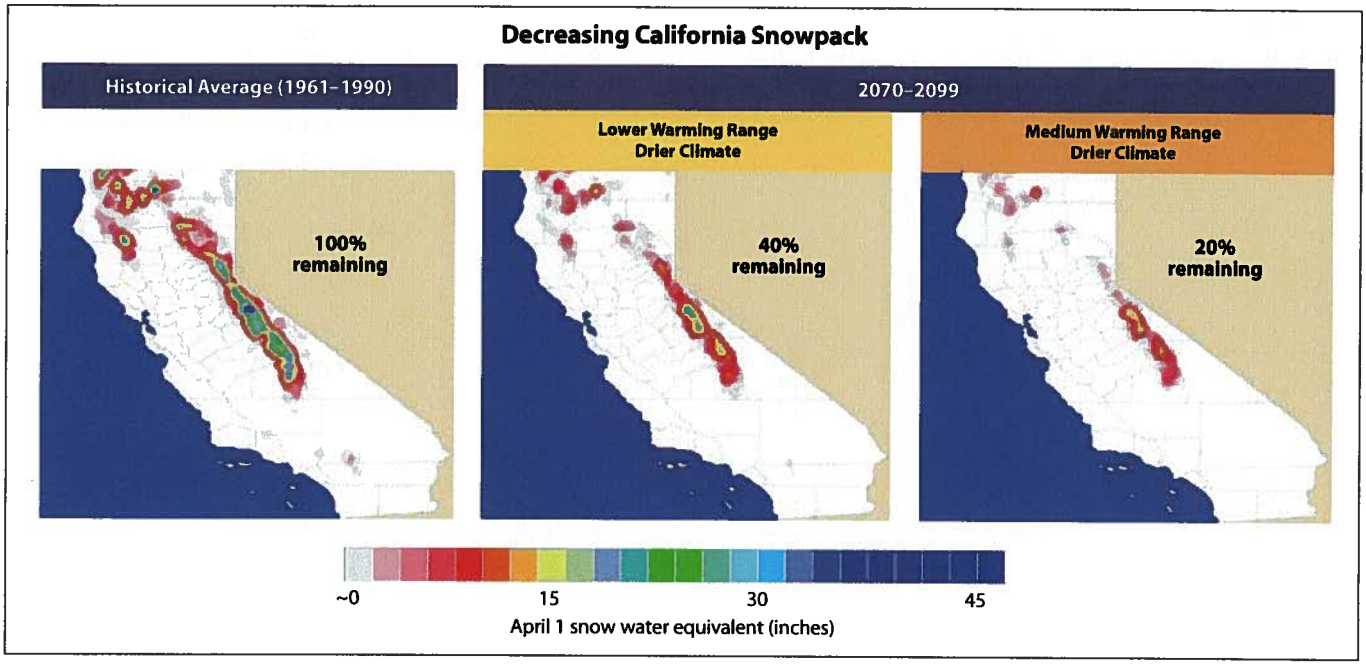
Most of California's precipitation falls in the northern part of the state during the winter while the greatest demand for water comes from users in the southern part of the state during the spring and summer. A vast network of man-made reservoirs and aqueducts capture and transport water throughout the state from northern California rivers and the Colorado River. The current distribution system relies on Sierra Nevada mountain snowpack to supply water during the dry spring and summer months. Rising temperatures, potentially compounded by decreases in precipitation, could severely reduce spring snowpack, increasing the risk of summer water shortages.

Decreasing Sierra Nevada Snowpack

If heat-trapping emissions continue unabated, more precipitation will fall as rain instead of snow, and the snow that does fall will melt earlier, reducing the Sierra Nevada spring snowpack by as much as 70 to 90 percent. How much snowpack will be lost depends in part on future precipitation patterns, the projections for which remain uncertain. However, even under wetter climate projections, the loss of snowpack would pose challenges to water managers, hamper hydropower generation, and nearly eliminate skiing and other snow-related recreational activities. If global warming emissions are significantly curbed and temperature increases are kept in the lower warming range, snowpack losses are expected to be only half as large as those expected if temperatures were to rise to the higher warming range.

Challenges in Securing Adequate Water Supplies

Continued global warming will increase pressure on California's water resources, which are already over-stretched by the demands of a growing



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economy and population. Decreasing snowmelt and spring stream flows coupled with increasing demand for water resulting from both a growing population and hotter climate could lead to increasing water shortages. By the end of the century, if temperatures rise to the medium warming range and precipitation decreases, late spring stream flow could decline by up to 30 percent. Agricultural areas could be hard hit, with California farmers losing as much as 25 percent of the water supply they need.

Water supplies are also at risk from rising sea levels. An influx of saltwater would degrade California's estuaries, wetlands, and groundwater aquifers. In particular, saltwater intrusion would threaten the quality and reliability of the major state fresh water supply that is pumped from the southern edge of the Sacramento/San Joaquin River Delta.

Coping with the most severe consequences of global warming would require major changes in water management and allocation systems. As more winter precipitation falls as rain instead of snow, water managers will have to balance the need to fill constructed reservoirs for water supply and the need to maintain reservoir space for winter flood control. Some additional storage could be developed; however, the economic and environmental costs would be high.

Left: Photodisc. Right: Corbis

Rising temperatures, potentially exacerbated by decreasing precipitation, could increase the risk of water shortages in urban and agricultural sectors.

Potential Reduction in Hydropower

Higher temperatures will likely increase electricity demand due to higher air conditioning use. Even if the population remained unchanged, toward the end of the century annual electricity demand could increase by as much as 20 percent if temperatures rise into the higher warming range. (Implementing aggressive efficiency measures could lower this estimate.)

At the same time, diminished snow melt flowing through dams will decrease the potential for hydropower production, which now comprises about 15 percent of California's in-state electricity production. If temperatures rise to the medium warming range and precipitation decreases by 10 to 20 percent, hydropower production may be reduced by up to 30 percent. However, future precipitation projections are quite uncertain so it is possible that precipitation may increase and expand hydropower generation.

Loss of Winter Recreation

Continued global warming will have widespread implications for winter tourism. Declines in Sierra Nevada snowpack would lead to later starting and earlier closing dates of the ski season. Toward the end of the century, if temperatures rise to the lower warming range, the ski season at lower and middle elevations could shorten by as much as a month. If temperatures reach the higher warming range and precipitation declines, there might be many years with insufficient snow for skiing and snowboarding.

Stephen McMillan

Agriculture

California is home to a $30 billion agriculture industry that employs more than one million workers. It is the largest and most diverse agriculture industry in the nation, producing more than 300 commodities including half the country's fruits and vegetables. Increased heat-trapping emissions are expected to cause widespread changes to this industry, reducing the quantity and quality of agricultural products statewide.

Although higher carbon dioxide levels can stimulate plant production and increase plant water-use efficiency, California farmers will face greater water demand for crops and a less reliable water supply as temperatures rise. Crop growth and development will change, as will the intensity and frequency of pest and disease outbreaks. Rising temperatures will likely aggravate ozone pollution, which makes plants more susceptible to disease and pests and interferes with plant growth.

To prepare for these changes, and to adapt to changes already under way, major efforts will be needed to move crops to new locations, respond to climate variability, and develop new cultivars and agricultural technologies. With adequate research and advance preparation, some of the consequences could be reduced.

Increasing Temperature

Plant growth tends to be slow at low temperatures, increasing with rising temperatures up to a threshold. However, faster growth can result in less-than-optimal development for many crops, so rising temperatures are likely to worsen the quantity and quality of yield for a number of California's agricultural products. Crops that are likely to be hard hit include:

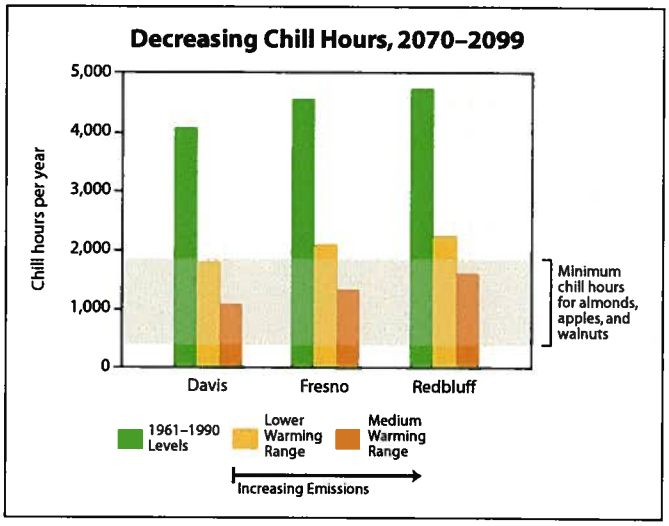
Wine Grapes

California is the nation's largest wine producer and the fourth-largest wine producer worldwide. High-quality wines produced throughout the Napa and Sonoma Valleys and along the northern and central coasts generate $3.2 billion in revenue each year. High temperatures during the growing season can cause premature ripening and reduce grape quality. Temperature increases are expected to have only modest effect on grape quality in most regions over the next few decades. However, toward the end of the century, wine grapes could ripen as much as one to two months earlier, which will affect grape quality in all but the coolest coastal locations (Mendocino and Monterey Counties).

IndexOpen

Fruits and Nuts

Many fruit and nut trees are particularly sensitive to temperature changes because of heat-accumulation limits and chill-hour requirements. Heat accumulation, which refers to the total hours during which temperatures reach between 45 and 95°F, is critical for fruit development. Rising temperatures could increase fruit development rates and decrease fruit size.



For example, peaches and nectarines developed and were harvested early in 2004 because of warm spring temperatures. The fruits were smaller than normal, which placed them in a lower quality category.

A minimum number of chill hours (hours during which temperatures drop below 45°F) is required for proper bud setting; too few hours can cause late or irregular bloom, decreasing fruit quality and subsequent marketable yield. California is currently classified as a moderate to high chill-hour region, but chill hours are diminishing in many areas of the state. If temperatures rise to the medium warming range, the number of chill hours in the entire Central Valley is expected to approach a critical threshold for some fruit trees.

Milk

California's $3 billion dairy industry supplies nearly one-fifth of the nation's milk products. High temperatures can stress dairy cows, reducing milk production. Production begins to decline at temperatures as low as 77°F and can drop substantially as temperatures climb above 90°F. Toward the end of the century, if temperatures rise to the higher warming range, milk production is expected to decrease by up to 20 percent. This is more

Increasing temperatures will likely decrease the quantity and quality of some agricultural commodities, such as certain varieties of fruit trees, wine grapes, and dairy products.

than twice the reduction expected if temperatures stay within or below the lower warming range.

Expanding Ranges of Agricultural Weeds

Noxious and invasive weeds currently infest more than 20 million acres of California farmland, costing hundreds of millions of dollars annually in control measures and lost productivity. Continued climate change will likely shift the ranges of existing invasive plants and weeds and alter competition patterns with native plants. Range expansion is expected in many species while range contractions are less likely in rapidly evolving species with significant populations already established. Should range contractions occur, it is likely that new or different weed species will fill the emerging gaps.

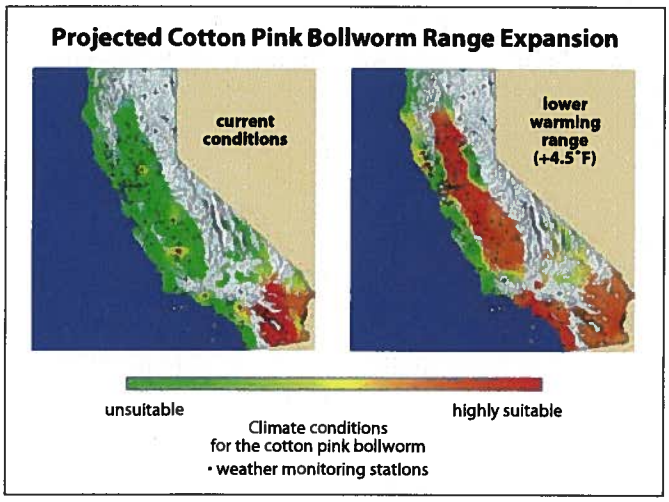
Increasing Threats from Pests and Pathogens

California farmers contend with a wide range of crop-damaging pests and pathogens. Continued climate change is likely to alter the abundance and types of many pests, lengthen pests' breeding season, and increase pathogen growth rates. For example, the pink bollworm, a common pest of cotton crops, is currently a problem only in southern desert valleys because it cannot survive winter frosts elsewhere in the state. However, if winter temperatures rise 3 to 4.5°F, the pink bollworm's range would likely expand northward, which could lead to substantial economic and ecological consequences for the state.

Temperature is not the only climatic influence on pests. For example, some insects are unable to cope in extreme drought, while others cannot survive in extremely wet conditions. Furthermore, while warming speeds up the lifecycles of many insects, suggesting that pest problems could increase, some insects may grow more slowly as elevated CO_2 levels decrease the protein content of the leaves on which they feed.

Multiple and Interacting Stresses

Although the effects on specific crops of individual factors (e.g., temperatures, pests, water supply) are increasingly well understood, trying to quantify interactions among these and other environmental factors is challenging. For example, the quality of certain grape varieties is expected to decline as temperatures rise. But the wine-grape industry also faces increasing risks from pests such as the glassy-winged sharpshooter, which transmits Pierce's disease. In 2002, this bacterial disease caused damage worth $13 million in Riverside County alone. The optimum temperature for growth of Pierce's disease is 82°F, so this disease is currently uncommon in the cooler northern and coastal regions of the state. However, with continued warming, these regions may face increased risk of the glassy-winged sharpshooter feeding on leaves and transmitting Pierce's disease.



U.S. Dept. of Agriculture

As temperatures rise, the climate is expected to become more favorable for the pink bollworm (above), a major cotton pest in southern California. The pink bollworm's geographic range is limited by winter frosts that kill over-wintering dormant larvae. As temperatures rise, winter frosts will decrease, greatly increasing the winter survival and subsequent spread of the pest throughout the state.

Forests and Landscapes

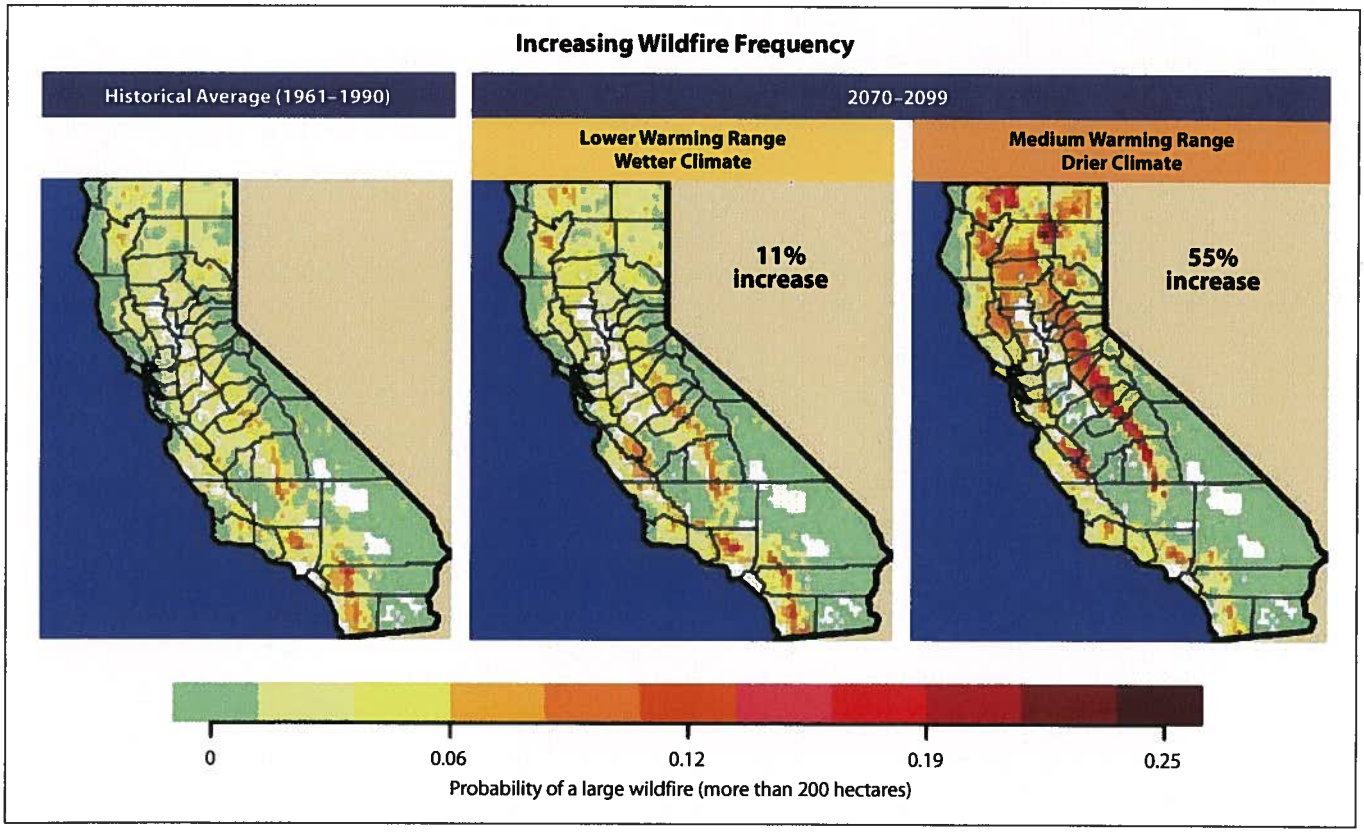
California is one of the most climatically and biologically diverse areas in the world, supporting thousands of plant and animal species. The state's burgeoning population and consequent impact on local landscapes is threatening much of this biological wealth. Global warming is expected to intensify this threat by increasing the risk of wildfire and altering the distribution and character of natural vegetation.

Increasing Wildfires

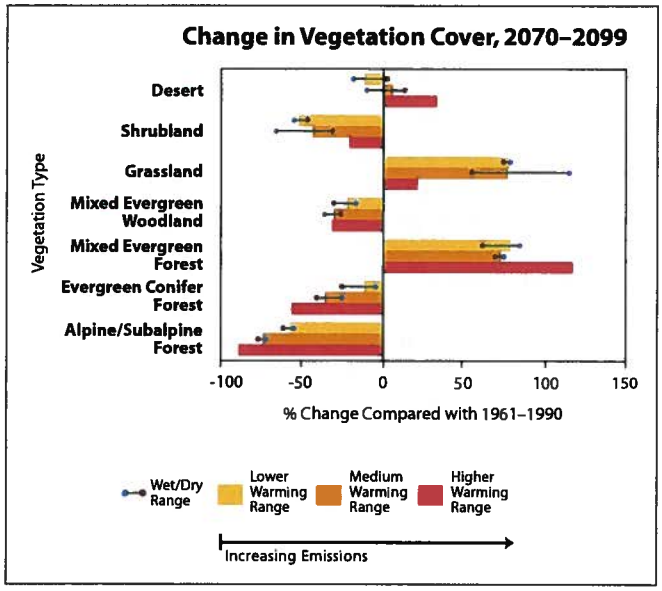
Fire is an important ecosystem disturbance. It promotes vegetation and wildlife diversity, releases nutrients into the soil, and eliminates heavy accumulation of underbrush that can fuel catastrophic fires. However, if temperatures rise into the medium warming range, the risk of large wildfires in California could increase by as much as 55 percent, which is almost twice the increase expected if temperatures stay in the lower warming range.

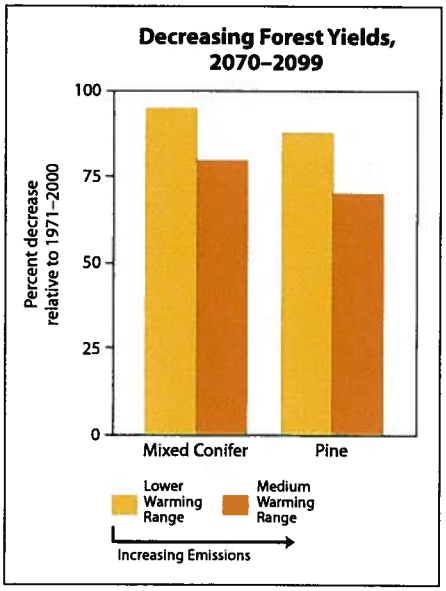
Because wildfire risk is determined by a combination of factors including precipitation, winds, temperature, and landscape and vegetation conditions, future risks will not be uniform throughout the state. In many regions, wildfire activity will depend critically on future precipitation patterns. For

Global warming threatens alpine and subalpine ecosystems, which have no place to move as temperatures rise.



Vegetation cover over the 21st century will depend on both temperature and precipitation. The lower and medium warming range bars reflect vegetation cover under a wetter climate (blue) and a drier climate (brown) projected in the different climate models. For the higher warming range, only a drier climate was considered.





example, if precipitation increases as temperatures rise, wildfires in the grasslands and chaparral ecosystems of southern California are expected to increase by approximately 30 percent toward the end of the century because more winter rain will stimulate the growth of more plant "fuel" available to burn in the fall. In contrast, a hotter, drier climate could promote up to 90 percent more northern California fires by the end of the century by drying out and increasing the flammability of forest vegetation.

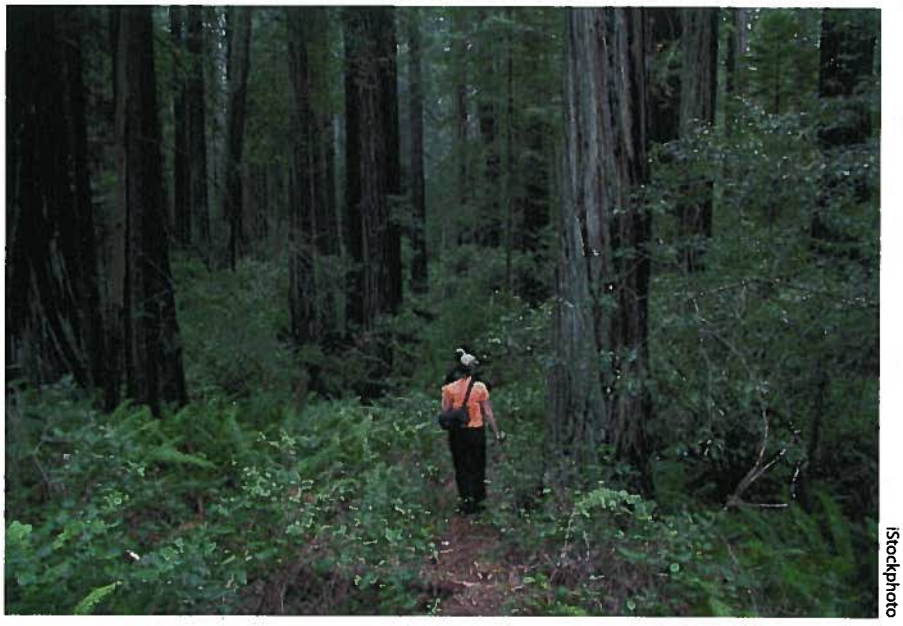
Shifting Vegetation

Land use and other changes resulting from economic development are altering natural habitats throughout the state. Continued global warming will intensify these pressures on the state's natural ecosystems and biological diversity. For example, in northern California, warmer temperatures are expected to shift dominant forest species from Douglas and White Fir to madrone and oaks. In inland regions, increases in fire frequency are expected to promote expansion of grasslands into current shrub and woodland areas. Alpine and subalpine ecosystems are among the most threatened in the state; plants suited to these regions have limited opportunity to migrate "up slope" and are expected to decline by as much as 60 to 80 percent by the end of the century as a result of increasing temperatures.

Declining Forest Productivity

Forestlands cover 45 percent of the state; 35 percent of this is commercial forests such as pine plantations. Recent projections suggest that continued global warming could adversely affect the health and productivity of California's forests. If average statewide temperatures rise to the medium warming range, the productivity of mixed conifer forests is expected to diminish by as much as 18 percent by the end of the century. Yield reductions from pine plantations are expected to be even more severe, with up to a 30 percent decrease by the end of the century.

The risk of large wildfires in California could increase by as much as 55 percent.



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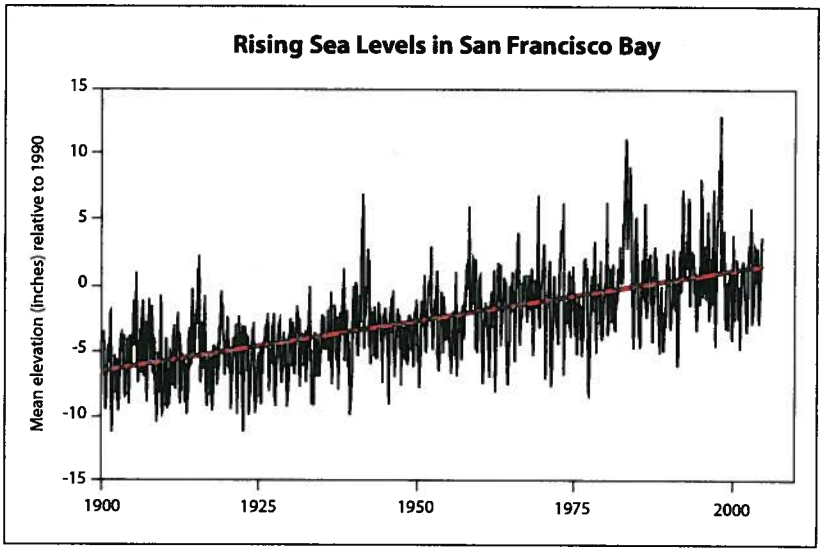
Rising Sea Levels

California's 1,100 miles of coastline are a major attraction for tourism, recreation, and other economic activity. The coast is also home to unique ecosystems that are among the world's most imperiled. As global warming continues, California's coastal regions will be increasingly threatened by rising sea levels, more intense coastal storms, and warmer water temperatures.

During the past century, sea levels along California's coast have risen about seven inches. If heat-trapping emissions continue unabated and temperatures rise into the higher warming range, sea level is expected to rise an additional 22 to 35 inches by the end of the century. Elevations of this magnitude would inundate coastal areas with salt water, accelerate coastal erosion, threaten vital levees and inland water systems, and disrupt wetlands and natural habitats.



Increasing Coastal Floods

The combination of increasingly severe winter storms, rising mean sea levels, and high tides is expected to cause more frequent and severe flooding, erosion, and damage to coastal structures. Many California coastal areas are at significant risk for flood damage. For example, the city of Santa Cruz is built on the 100-year floodplain and is only 20 feet above sea level. Although levees have been built to contain the 100-year flood, a 12-inch increase in sea levels (projected for the medium warming range of temperatures) would mean storm-surge-induced flood events at the 100-year level would likely occur once every 10 years.

Flooding can create significant damage and enormous financial losses. Despite extensive engineering efforts, major floods have repeatedly breached levees that protect freshwater supplies and islands in the San Francisco Bay Delta as well as fragile marine estuaries and wetlands throughout the

Robert A. Eppleti/CA Governor's Office of Emergency Services

Rising sea levels and more intense storm surges could increase the risk for coastal flooding.

Sea levels could rise up to three feet by the end of the century, accelerating coastal erosion, threating vital levees, and disrupting wetlands.

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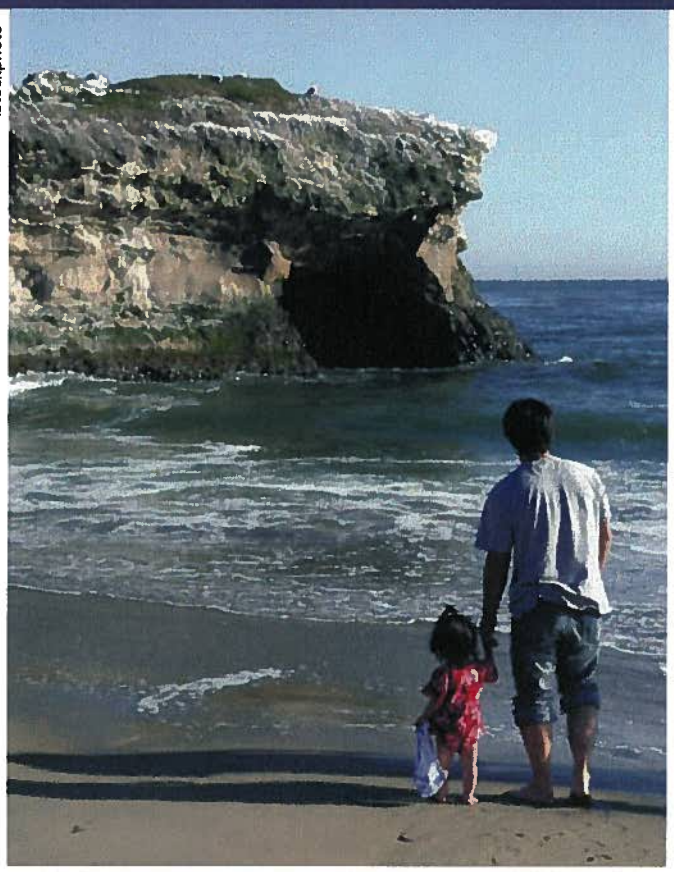
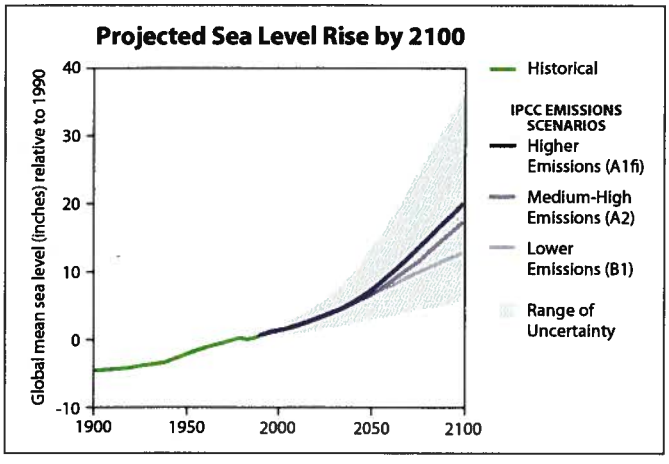


Many California beaches are threatened from rising sea levels and increased erosion, an expected consequence of continued global warming.

state. Continued sea level rise will further increase vulnerability to levee failures. Some of the most extreme flooding during the past few decades has occurred during El Niño winters, when warmer waters fuel more intense storms. During the winters of 1982–1983 and 1997–1998, for example, abnormally high seas and storm surges caused millions of dollars' worth of damage in the San Francisco Bay area. Highways were flooded as six-foot waves crashed over waterfront bulkheads, and valuable coastal real estate was destroyed.

Continued global warming will require major changes in flood management. In many regions such as the Central Valley, where urbanization and limited river channel capacity already exacerbate rising flood risks, flood damage and flood control costs could amount to several billion dollars.



Shrinking Beaches

Many of California's beaches may shrink in the future because of rising seas and increased erosion from winter storms. Currently, many beaches are protected from erosion through manmade sand replenishment (or "nourishment") programs, which bring in sand from outside sources to replace the diminishing supply of natural sand. In fact, many of the wide sandy beaches in southern California around Santa Monica, Venice, and Newport Beach were created and are maintained entirely by sand nourishment programs. As sea levels rise, increasing volumes of replacement sand will be needed to maintain current beach width and quality. California beach nourishment programs currently cost millions of dollars each year. As global warming continues, the costs of beach nourishment programs will rise, and in some regions beach replenishment may no longer be viable.

Multiple Causes of Coastal Flooding

Several factors play a role in sea level and coastal flooding, including tides, waves temperature, and storm activity. Sea levels fluctuate daily, monthly, and seasonally; the highest tides occur in winter and in summer, during new and full moons. Sea levels often rise even higher during El Niño winters, when the Eastern Pacific Ocean is warmer than usual and westerly wind patterns are strengthened.

Coastal flooding usually occurs during winter storms, which bring strong winds and high waves. Storm winds tend to raise water levels along the coast and produce high waves at the same time, compounding the risk of damaging waves—a doubling of wave height is equivalent to a four-fold increase in wave energy. When these factors coincide with high tides, the chances for coastal damage are greatly heightened.

As sea levels rise, flood stages in the Sacramento/San Joaquin Delta of the San Francisco Bay estuary may also rise, putting increasing pressure on Delta levees. This threat may be particularly significant because recent estimates indicate the additional force exerted upon the levees is equivalent to the square of the water level rise. Estimates using historical observations and climate model projections suggest that extreme high water levels in the Bay and Delta will increase markedly if sea level rises above its historical rate. These extremes are most likely to occur during storm events, leading to more severe damage from waves and floods.

Top left & right: Photos.com. Bottom: CA Fuel Cell Partnership

Cleaner energy and vehicle technologies can help California reduce global warming emissions, improve air quality, and protect public health.

Managing Global Warming

Continued global warming will have widespread and significant impacts on the Golden State. Solutions are available today to reduce emissions and minimize these impacts.

The projections presented in this analysis suggest that many of the most severe consequences that are expected from the medium and higher warming ranges could be avoided if heat-trapping emissions can be reduced to levels that will hold temperature increases at or below the lower warming range (i.e., an increase of no more than 5.5°F). However, even if emissions are substantially reduced, research indicates that some climatic changes are unavoidable. Although not the solution to global warming, plans to cope with these changes are essential.

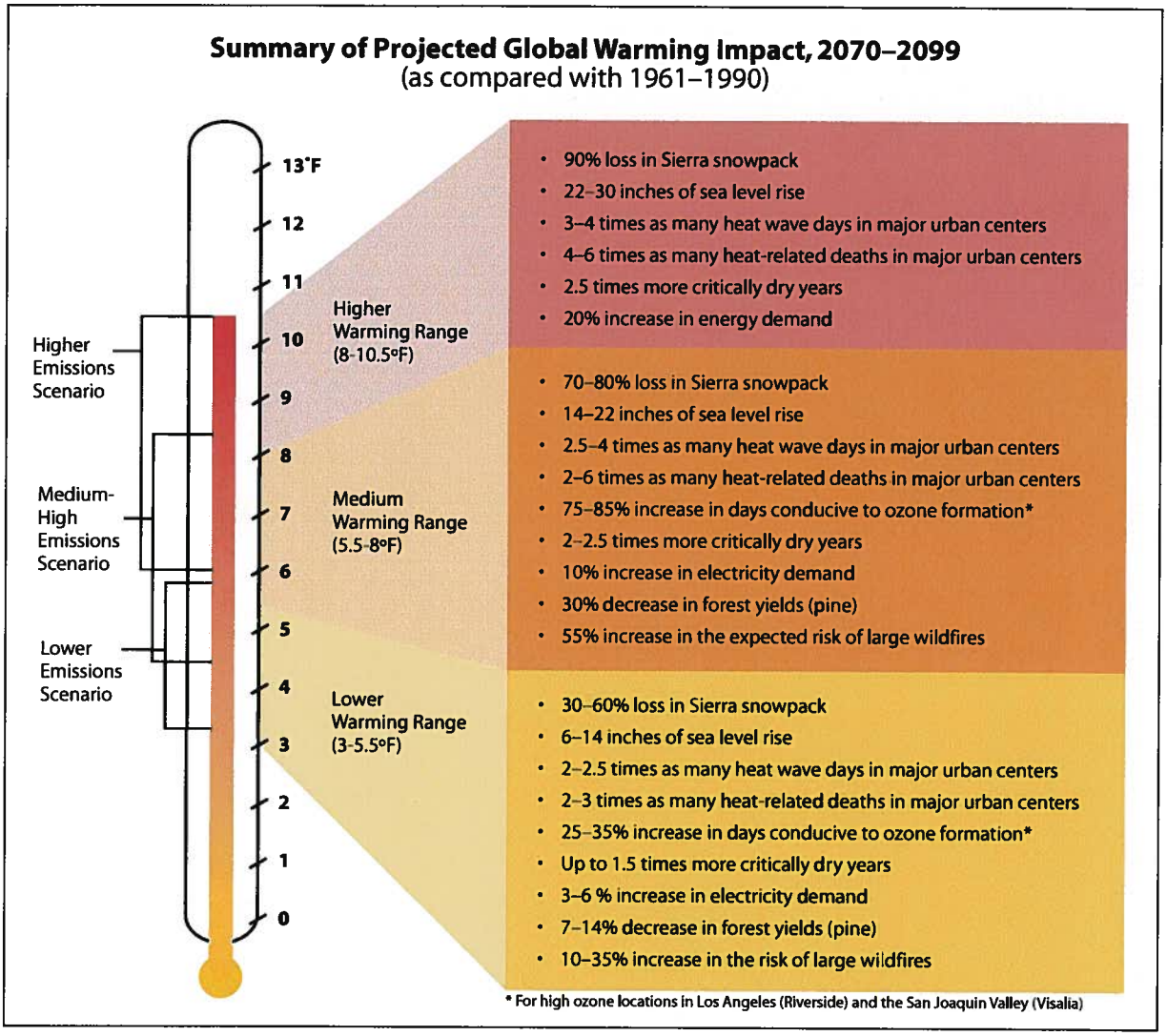
California's actions can drive global progress to address global warming.

Reducing Heat-Trapping Emissions

Reducing heat-trapping emissions is the most important way to slow the rate of global warming. On June 1, 2005, Governor Arnold Schwarzenegger signed an executive order (#S-3-05) that sets goals for significantly lowering the state's share of global warming pollution. The executive order calls for a reduction in heat-trapping emissions to 1990 levels by 2020 and for an 80 percent emissions reduction below 1990 levels by 2050. These emission reduction targets will help stimulate technological innovation needed to help transition to more efficient and renewable transportation and energy systems.

Coping with Unavoidable Climatic Changes

Because global warming is already upon us, and some amount of additional warming is inevitable, we must prepare for the changes that are already under way.



Preparing for these unavoidable changes will require minimizing further stresses on sensitive ecosystems and implementing management practices that integrate climate risks into long-term planning strategies.

California's Leadership

California has been a leader in both the science of climate change and in identifying solutions. The California Climate Change Center is one of the first—and perhaps the only—state-sponsored research institution in the nation dedicated to climate change research, and other state agencies such as the Air Resources Board support similar research. Continuing this strong research agenda is critical for developing effective strategies for addressing global warming in California.

The state has also been at the forefront of efforts to reduce heat-trapping emissions, passing precedent-setting policies such as aggressive standards for tailpipe emissions, renewable energy, and energy efficiency. However, existing policies are not likely to be sufficient to meet the ambitious emission reduction goals set by the governor. To meet these ambitious goals California will need to build on its legacy of environmental leadership and develop new strategies and technologies to reduce emissions.

By reducing heat-trapping emissions, severe consequences can be avoided.

California alone cannot stabilize the climate. However, the state's actions can drive global progress. If the industrialized world were to follow the emission reduction targets established in California's executive order, and industrializing nations reduced emissions according to the lower emissions path (B1) presented in this analysis, we would be on track to keep temperatures from rising to the medium or higher (and possibly even the lower) warming ranges and thus avoid the most severe consequences of global warming.

The full text of the Climate Scenarios analysis overview report, and the core scientific papers that comprise this analysis, are online at *www.climatechange.ca.gov*. The scientists that participated in this effort are:

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